

## Chapter 7. Diagnostic Evaluation and Medical Management of Children With Blood Lead Levels $\geq 20$ $\mu\text{g/dL}$

### *Summary*

*Children with blood lead levels  $\geq 20$   $\mu\text{g/dL}$  need complete medical evaluations.*

*Several pharmacologic agents can reduce blood lead levels; however, the most important factor is reducing the child's exposure to lead.*

*Research and new developments may change many aspects of the medical management of poisoned children.*

Children with blood lead levels between 10  $\mu\text{g/dL}$  and 19  $\mu\text{g/dL}$  and their siblings need followup and repeat screening as described in previous chapters. They do not, however, need medical evaluation as described in this chapter.

The cornerstones of clinical management are careful clinical and laboratory surveillance of the child, medical treatment when indicated, and eradication of controllable sources of environmental lead. **The most important factor in case management is to reduce the child's exposure to lead.**

All children with confirmed venous blood lead levels  $\geq 20$   $\mu\text{g/dL}$  require medical evaluation. The urgency of further medical evaluation depends on the blood lead level and whether symptoms are present.

The decision to institute medical management should virtually always be made on the basis of a venous blood lead measurement. No other screening test can be considered diagnostic. If the first evaluation was made on capillary blood, a confirmatory venous blood lead level must be done. Even if the first diagnostic measurement was on venous blood, it is preferable to retest before starting chelation therapy. For children with blood lead levels  $\geq 70$   $\mu\text{g/dL}$  or clinical symptoms of lead poisoning, chelation should not be postponed while awaiting results of the repeat test.

### **SYMPTOMS OF LEAD POISONING**

Symptomatic lead poisoning is a medical emergency.

**Symptoms of lead poisoning in a child with an elevated blood lead level constitute a medical emergency, and the child should be hospitalized.** Symptoms, which can mimic several other pediatric disorders, must be looked for so they are not missed (Piomelli et al., 1984).

Acute lead encephalopathy is characterized by some or all of these symptoms: coma, seizures, bizarre behavior, ataxia, apathy, incoordination, vomiting, alteration in the state of consciousness, and subtle loss of recently acquired skills. Any one or a mixture of these symptoms, associated with an elevated blood lead level, is an acute medical emergency. Lead encephalopathy is almost always associated with a blood lead level exceeding 100  $\mu\text{g}/\text{dL}$ , although, occasionally, it has been reported at blood lead levels as low as 70  $\mu\text{g}/\text{dL}$ . Even when identified and promptly treated, severe and permanent brain damage may result in 70%-80% of children with lead encephalopathy (Perlstein and Attala, 1966). Children with symptomatic lead poisoning with or without encephalopathy represent an acute medical emergency. **The possibility of lead encephalopathy should be considered in the differential diagnosis of children presenting with coma and convulsions of unknown etiology.**

Except for coma and seizures, symptomatic lead poisoning without encephalopathy is characterized by symptoms similar to those of lead encephalopathy. Symptomatic lead poisoning without encephalopathy is characterized by one or a combination of these symptoms: decrease in play activity, lethargy, anorexia, sporadic vomiting, intermittent abdominal pain, and constipation. These symptoms are usually associated with a blood lead levels of at least 70  $\mu\text{g}/\text{dL}$ , although occasionally cases have been associated with levels as low as 50  $\mu\text{g}/\text{dL}$ . If the blood lead level is below 50  $\mu\text{g}/\text{dL}$ , other causes of the symptoms should be sought. **Since acute lead encephalopathy may develop in any symptomatic child, treatment and supportive measures must be started immediately on an emergency basis.**

## EVALUATION OF THE CHILD WITH A BLOOD LEAD LEVEL $\geq 20$ $\mu\text{g}/\text{dL}$

Take a careful history and do a physical examination.  
Include evaluation of the child's iron status and other special diagnostic tests.

### History and Physical Examination

A child with a blood lead level  $\geq 20$   $\mu\text{g}/\text{dL}$  should have a pediatric evaluation, whether or not symptoms are present.

Special attention should be given to:

1. A detailed history, including the presence or absence of clinical symptoms, child's mouthing activities, the existence of pica, nutritional status (especially iron and calcium intake), dietary habits, family history of lead poisoning, potential sources of lead exposure (including exposure due to home renovation), and previous blood lead measurements.
2. Detailed environmental and occupational histories of adults in the household or other places the child spends a lot of time.
3. The physical examination, with particular attention to the neurologic examination and psychosocial and language development. A neurobehavioral assessment may be useful in children receiving chelation therapy both at the time of diagnosis and as the child approaches school age. Findings of language delay or other problems can prompt referral to appropriate programs.

4. Evaluation of iron status using measurement of iron and total iron binding capacity or of ferritin.

## **Iron Status and Special Tests**

### ***1. Tests for Iron Deficiency***

**Because iron deficiency can enhance lead absorption and toxicity and often coexists with it, all children with blood lead levels  $\geq 20$   $\mu\text{g/dL}$  should be tested for iron deficiency.** Measurements of hemoglobin, hematocrit, and reticulocytes are not adequately sensitive, and erythrocyte protoporphyrin (EP) is not specific enough to diagnose iron deficiency (although EP can be used to screen for iron deficiency).

**Serum iron and iron binding capacity (transferrin saturation) and ferritin** are the most sensitive indicators of iron status. An abnormally low ratio of serum iron to iron binding capacity (transferrin saturation) of 0.2 is consistent with iron deficiency. The serum ferritin level, however, is the most definitive and accurate indication of overall iron status, although it is an acute phase reactant and may be falsely elevated in sick children; a value  $\leq 12$   $\mu\text{g/dL}$  indicates iron deficiency. Although all iron deficient children should receive treatment for this condition, the treatment should not be started until after chelation is completed in children receiving dimercaprol (BAL).

### ***2. EP Level***

An elevated EP level indicates impairment of the heme biosynthetic pathway. EP levels are sensitive screening tests for iron deficiency, and iron status should be assessed in any child with an elevated EP level (that is,  $\geq 35$   $\mu\text{g/dL}$  when standardized using  $241 \text{ L cm}^{-1} \text{ mmol}^{-1}$ ,  $\geq 28$   $\mu\text{g/dL}$  when standardized using  $297 \text{ L cm}^{-1} \text{ mmol}^{-1}$ , or  $\geq 70$   $\mu\text{mol/mol}$  when measured in  $\mu\text{mol/mol}$  units).

Because EP levels take about 2 weeks to increase, EP levels may provide an indication of the duration of lead exposure (Chisolm, 1982; Chisolm, personal communication). Similarly, monitoring the EP level after medical and environmental interventions for poisoned children may be useful. If exposure to lead has ceased, EP values elevated because of lead poisoning decline slowly over several weeks or months (Piomelli et al., 1984). A progressive decline in EP concentrations indicates that combined medical and environmental case management is proceeding efficaciously.

### ***3. Edetate Disodium Calcium (CaNa<sub>2</sub>EDTA) Provocative Chelation Test***

The mobilization test is used to determine whether a child with an initial confirmatory blood lead level of 25 to 44  $\mu\text{g/dL}$  will respond to chelation therapy with a brisk lead diuresis (Piomelli et al., 1984; Markowitz and Rosen, 1991). Because of the cost and staff time needed for quantitative urine collection, this test is used only in selected medical centers where large numbers of lead-poisoned children are treated. Children whose blood lead levels are  $\geq 45$   $\mu\text{g/dL}$  should not receive a provocative chelation test; they should be referred for appropriate chelation therapy immediately.

The outcome of the provocative chelation test is determined not by a decrease in the blood lead level but by the amount of lead excreted per dose of CaNa<sub>2</sub>EDTA given. This ratio correlates well with blood lead levels. In one study, almost all children with blood lead levels 45  $\mu\text{g/dL}$  had positive provocative tests, 76% of the children with blood lead levels 35 to 44  $\mu\text{g/dL}$

had positive test results, and 35% of the children with blood lead levels 25 to 34  $\mu\text{g}/\text{dL}$  had positive test results (Markowitz and Rosen, 1991). This test should not be done until the child is iron replete, since iron status may affect the outcome of the test (Markowitz et al., 1990).

**Conducting a  $\text{CaNa}_2\text{EDTA}$  Provocative Chelation Test.** First, a repeated baseline blood lead level must be obtained. The patient is asked to empty the bladder, and then  $\text{CaNa}_2\text{EDTA}$  is administered at a dose of  $500 \text{ mg}/\text{m}^2$  in 5% dextrose infused **over 1 hour**. (A somewhat painful but practical alternative is to administer intramuscularly the same dose mixed with procaine so that the final concentration of procaine is 0.5%.) All urine must be collected with lead-free equipment over the next 8 hours. (An 8 hour mobilization test has been shown to be as reliable as a 24-hour mobilization test (Markowitz and Rosen, 1984).) An 8-hour test can be accomplished on an out-patient basis, but the patient should not leave the clinic during this test.) In the laboratory, the urine volume should be carefully measured and stored at  $20^\circ\text{C}$  until the lead concentration is measured. Extreme care must be taken to ensure that lead-free equipment is used.

The use of lead-free apparatus for urine collection is mandatory. Special lead-free collection apparatus must be used if valid test results are to be obtained. The laboratory that will perform the analysis should supply the proper collection apparatus. Preferably, urine should be voided directly into polyethylene or polypropylene bottles that have been cleaned by the usual procedures, then washed in nitric acid, and thoroughly rinsed with deionized, distilled water. For children who are not toilet trained, plastic pediatric urine collectors can be used. Urine collected in this manner should be transferred directly to the urine collection bottles.

**Interpretation of a  $\text{CaNa}_2\text{EDTA}$  Provocative Chelation Test.** To obtain the total lead excretion in micrograms, the concentration of lead in the urine (in micrograms per milliliter) is multiplied by the total urinary volume (in milliliters). The total urinary excretion of lead (micrograms) is divided by the amount of  $\text{CaNa}_2\text{EDTA}$  given (milligrams) to obtain the lead excretion ratio:

$$\frac{\text{Lead excreted } (\mu\text{g})}{\text{CaNa}_2\text{EDTA given (mg)}}$$

An 8-hour  $\text{CaNa}_2\text{EDTA}$  chelation provocative test is considered positive if the lead excretion ratio is  $>0.6$  (Markowitz and Rosen, 1991). Some clinicians use a cutoff of 0.5 for the lead excretion ratio (Weinberger et al., 1987). Children with blood lead levels 25 to  $44 \mu\text{g}/\text{dL}$  and positive chelation test results should undergo a 5-day course of chelation.

Regardless of age, all children with elevated blood lead values and negative provocative chelation results should have blood lead levels measured monthly. If the elevation in blood lead values persists, the  $\text{CaNa}_2\text{EDTA}$  provocative test can be repeated every 1 to 3 months and interpreted according to the above guidelines.

#### ***4. Radiologic Examination of the Abdomen***

Radiologic examination of the abdomen (flat plate) may show radiopaque foreign material if the material has been ingested during the preceding 24 to 36 hours. Neither negative nor positive xray results are diagnostic or definitive. A flat plate of the abdomen may, however, provide information about the source of lead if paint chips or other lead objects are found.

#### ***5. Radiologic Examination of the Long Bones***

Xrays of the long bones are unreliable for diagnosing acute lead poisoning, and they should not be obtained on a routine basis. They may provide some indication of whether lead poisoning

has occurred in the past or has been ongoing for a length of time, and this may occasionally be important. Lines of increased density in the metaphyseal plate of the distal femur, proximal tibia, and fibula may be caused by lead which has disrupted the metabolism of bone matrix. Although these lines are sometimes called lead lines, they are areas of increased mineralization or calcification and not xray shadows of deposited lead.

The following tests are **NOT** indicated for the diagnosis or clinical management of lead poisoning:

**1. Microscopic Examination of Red Cells for Basophilic Stippling**

Since basophilic stippling is not always found in severe lead poisoning and is insensitive to lesser degrees of lead poisoning, it is not useful in diagnosis.

**2. Tests of Hair and Fingernails for Lead Levels**

The levels of lead in hair or fingernails do not correlate well with blood lead levels, except in extreme cases of symptomatic lead poisoning; therefore, these tests are not useful in diagnosis. Children should never receive chelating agents on the basis of analyses of lead levels in hair or fingernails.

**PHARMACOLOGY OF CHELATING AGENTS**

<b>Chelating Agents Used In Treating Children With Lead Poisoning</b>			
<b>Product Name</b>	<b>Generic Name</b>	<b>Chemical Name</b>	<b>Abbreviation</b>
Calcium Disodium Versenate	Edetate disodium calcium	Calcium disodium ethylenediamine tetraacetate	CaNa <sub>2</sub> EDTA
BAL in Oil	Dimercaprol	2,3-dimercapto-1-propanol	BAL
Cuprimine	D-penicillamine	3-mercapto-D-valine	D-penicillamine
Chemet	Succimer	Meso 2,3-dimercaptosuccinic acid	DMSA

Several drugs are used in the treatment of lead poisoning. These drugs, capable of binding or chelating lead, deplete the soft and hard (skeletal) tissues of lead and thus reduce its acute toxicity (Chisolm, 1968; Markowitz and Rosen, 1984; Piomelli et al., 1984; Rosen et al., in press). All drugs have potential side effects and must be used with caution (Piomelli et al., 1984). The basic pharmacologic characteristics of the various drugs are described below.

**BAL**

**Mechanism of action.** Two molecules of dimercaprol (BAL) combine with one atom of heavy metal to form a stable complex. BAL enhances fecal and urinary excretion of lead and diffuses well into erythrocytes. Because it is predominantly excreted in bile, BAL can be administered in the presence of renal impairment (Chisolm, 1968).

**Route of administration and dosage.** BAL is available only in peanut oil for intramuscular administration. It is usually given every 4 hours, although it may be given every 8 hours; dosages are discussed starting on page 59.

**Precautions and Toxicity.** For patients with glucose-6-phosphate dehydrogenase deficiency (G-6-PD), some clinicians recommend that BAL should be used only in life-threatening situations because it may induce hemolysis. Medicinal iron should never be administered during BAL therapy, because the combination of iron and BAL has been implicated in serious reactions. If iron deficiency coexists, it should not be treated until after BAL therapy has been completed. In cases of extreme anemia, blood transfusions are preferable.

Between 30% and 50% of patients who receive BAL will experience side effects. Mild febrile reactions and transient elevations of hepatic transaminases may be observed. Other minor adverse effects include, in order of frequency, nausea and occasional vomiting, headache, mild conjunctivitis, lacrimation, rhinorrhea, and salivation. Most side effects are transient and rapidly subside as the drug is metabolized and excreted. Intravenous hydration coupled with restricting oral intake can circumvent, in large part, gastrointestinal distress.

**BAL should not be used for children who are allergic to peanuts or peanut products.**

## **CaNa<sub>2</sub>EDTA**

*Only CaNa<sub>2</sub>EDTA can be used for treating children with lead poisoning. Na<sub>2</sub>EDTA (disodium edetate) should never be used for treating children with lead poisoning because it will induce tetany and possibly fatal hypocalcemia.*

**Mechanism of action.** CaNa<sub>2</sub>EDTA increases urinary lead excretion twentyfold to fiftyfold. CaNa<sub>2</sub>EDTA removes lead from the extracellular compartment only, because it does not enter cells (Osterloh and Becker, 1986).

**Route of administration and dosage.** The preferred route for administration of CaNa<sub>2</sub>EDTA is intravenous. CaNa<sub>2</sub>EDTA must be diluted to a concentration of <0.5% either in dextrose and water or in 0.9% saline solution. It can be given as a continuous infusion or it can be given in two divided doses a day through a heparin lock over 30 to 60 minutes. CaNa<sub>2</sub>EDTA causes extreme pain when administered intramuscularly; therefore, when given by this route, it should be mixed with procaine so that the final concentration of procaine is 0.5%. CaNa<sub>2</sub>EDTA should never be given orally because it enhances absorption of lead from the gastrointestinal tract.

Dosages vary by situation and are detailed starting on page 59. Individual courses should be limited to 5 days and repeated courses should be given at a minimum of 2- to 5-day intervals. Particularly when CaNa<sub>2</sub>EDTA is given on an outpatient basis, some clinicians use sequential 3-day courses of treatment.

**Precautions and Toxicity.** During chelation therapy with CaNa<sub>2</sub>EDTA, urine output, urine sediment, blood urea nitrogen (BUN), serum creatinine, and hepatocellular enzyme levels must be carefully monitored. The appearance of protein and formed elements in urinary sediment, and rising BUN and serum creatinine values reflect impending renal failure—the serious toxicity associated with inappropriately excessive or prolonged administration of CaNa<sub>2</sub>EDTA. Liver transaminases may increase by the fifth day of therapy, but return to pretreatment levels within a week after treatment has ended.

When CaNa<sub>2</sub>EDTA is used alone without concomitant BAL therapy, it may aggravate symptoms in patients with very high blood lead levels. Therefore, it should be used in conjunction with BAL when the blood lead level is  $\geq 70$   $\mu\text{g/dL}$  or overt clinical symptoms of lead poisoning are present. In such cases, the first dose of BAL should always precede the first dose of CaNa<sub>2</sub>EDTA by at least 4 hours.

The kidney is the principal site of potential toxicity. Renal toxicity is dose related, reversible, and rarely (if ever) occurs at doses  $<1500 \text{ mg/m}^2$  when the patient is adequately hydrated.  $\text{CaNa}_2\text{EDTA}$  must never be given in the absence of an adequate urine flow (Piomelli et al., 1984).

### **D-penicillamine**

The Food and Drug Administration (FDA) has approved D-penicillamine for the treatment of Wilson's disease, cystinuria, and severe, active rheumatoid arthritis. Although not approved for this use, it is used in some centers for treating lead poisoning. Until the recent approval of succimer, it was the only commercially available oral chelating agent. It can be given over a long period (weeks to months). D-penicillamine has been used mainly for children with blood lead levels  $<45 \text{ } \mu\text{g/dL}$ .

**Mechanism of action.** D-penicillamine enhances urinary excretion of lead, although not as effectively as  $\text{CaNa}_2\text{EDTA}$ . Its specific mechanism and site of action are not well understood.

**Route of administration and dosage.** D-penicillamine is administered orally. It is available in capsules or tablets (125 mg and 250 mg). These capsules can be opened and suspended in liquid, if necessary. The usual dose is 25 to 35 mg/kg/day in divided doses. Side effects can be minimized, to an extent, by starting with a small dose and increasing it gradually, monitoring all the time for side effects. For example, 25% of the desired final dose could be given in week 1, 50% in week 2, and the full dose by week 3.

**Precautions and Toxicity.** Toxic side effects (albeit minor in most cases) occur in as many as 33% of patients given the drug (Shannon et al., 1988). The main side effects of D-penicillamine are reactions resembling those of penicillin sensitivity, including rashes, leukopenia, thrombocytopenia, hematuria, proteinuria, hepatocellular enzyme elevations, and eosinophilia. Anorexia, nausea, and vomiting are infrequent. Of most concern, however, are isolated reports of nephrotoxicity, possibly from hypersensitivity reactions. For these reasons, patients should be carefully and frequently monitored for clinically obvious side effects, and frequent blood counts, urinalyses, and renal function tests should be performed. In particular, blood counts and urinalyses should be done on day 1, day 14, day 28, and monthly thereafter. If the absolute neutrophil count falls to  $<1500/\mu\text{L}$ , the count should be rechecked immediately, and treatment should be stopped if it falls to  $<1200/\mu\text{L}$ . D-penicillamine should not be given on an outpatient basis if exposure to lead is continuing or the physician has doubts about compliance with the therapeutic regimen.

**D-penicillamine should not be administered to patients with known penicillin allergy.**

### **Succimer**

The FDA approved succimer in January, 1991 for treating children with blood lead levels  $>45 \text{ } \mu\text{g/dL}$ . Succimer appears to be an effective oral chelating agent. Its selectivity for lead is high, whereas its ability to chelate essential trace metals is low. Although its use to date has been limited, succimer appears to have promising potential, and a broader range of clinical research studies in children are being undertaken.

Succimer is chemically similar to BAL but is more water soluble, has a high therapeutic index, and is absorbed from the gastrointestinal tract (Aposhian and Aposhian, 1990). It is

effective when given orally and produces a lead diuresis comparable to that produced by  $\text{CaNa}_2\text{EDTA}$  (Chisolm, 1990). This diuresis lowers blood lead levels and reverses the biochemical toxicity of lead, as indicated by normalization of circulating aminolevulinic acid dehydrase levels (Graziano et al., 1988). Succimer is not indicated for prophylaxis of lead poisoning in a lead-containing environment. **As with all chelating agents, succimer should only be given to children who reside in environments free of lead during and after treatment.**

**Mechanism of Action.** Succimer appears to be more specific for lead than the most commonly used chelating agent,  $\text{CaNa}_2\text{EDTA}$ ; the urinary loss of essential trace elements (for example, zinc) appears to be considerably less with succimer than with  $\text{CaNa}_2\text{EDTA}$  (Aposhian and Aposhian, 1990). The site of lead chelation by succimer is not known.

**Route of Administration and Dosage.** Succimer is administered orally. It is available in 100 mg capsules. The recommended initial dose is  $350 \text{ mg/m}^2$  (10 mg/kg) every 8 hours for 5 days, followed by  $350 \text{ mg/m}^2$  (10 mg/kg) every 12 hours for 14 days. A course of treatment, therefore, lasts 19 days. If more courses are needed, a minimum of 2 weeks between courses is preferred, unless blood lead levels indicate the need for immediate retreatment. These doses may be modified as more experience is gained in using succimer.

Patients who have received therapeutic courses of  $\text{CaNa}_2\text{EDTA}$  with or without BAL may use succimer for subsequent treatment after an interval of 4 weeks. Data on the concomitant use of succimer and  $\text{CaNa}_2\text{EDTA}$  with or without BAL are not available, and such use is not recommended.

If young children cannot swallow capsules, succimer can be administered by separating the capsule and sprinkling the medicated beads on a small amount of soft food or by putting them on a spoon and following with a fruit drink. Data are not available on how stable succimer is when it is suspended in soft foods for prolonged periods of time; succimer should be mixed with soft foods immediately before being given to the child.

**Precautions and Toxicity.** To date, toxicity due to succimer (transient elevations in hepatic enzyme activities) appears to be minimal (Graziano et al., 1988). The most common adverse effects reported in clinical trials in children and adults were primarily gastrointestinal and included nausea, vomiting, diarrhea, and appetite loss. Rashes, some necessitating discontinuation of therapy, have been reported for about 4% of patients. **Though succimer holds considerable promise for the outpatient management of lead poisoning, clinical experience with succimer is limited.** Consequently, the full spectrum and incidence of adverse reactions, including the possibility of hypersensitivity or idiosyncratic reactions, have not been determined.

If succimer is used, the following precautions must be taken:

1. Monitor for side effects (especially effects on liver transaminases), the rapidity of the initial decrease in blood lead levels, and the course of the rebound in blood lead levels once treatment has ended.
2. **Succimer, like other chelators, is not a substitute for effective and rapid environmental interventions.** Use succimer as part of an integrated environmental and medical approach to treating patients with lead poisoning.
3. Do not give succimer (or any other chelating agent) in situations where high dose lead sources are available to the child. In rats, gastrointestinal absorption of lead and whole body lead retention were reduced by a single oral dose of succimer (Kapoor et al., 1989). The potential for enhancing human lead absorption from the gastrointestinal tract during the use of succimer is under study.

4. Children with blood lead levels  $>45 \mu\text{g/dL}$  who are being treated with succimer, should, if possible, be hospitalized until their blood lead levels fall below  $45 \mu\text{g/dL}$  and the lead hazards in their homes are abated or alternative lead hazard-free housing has been identified.
5. Children with blood lead levels  $\geq 70 \mu\text{g/dL}$  should be immediately hospitalized. The decision to treat such children with succimer instead of  $\text{CaNa}_2\text{EDTA}$  and BAL should be made with the understanding that experience with using succimer in children with these blood lead levels is limited.

## **TREATMENT GUIDELINES FOR CHILDREN WITH BLOOD LEAD LEVELS $\geq 20 \mu\text{g/dL}$**

The most important factor in managing childhood lead poisoning is reducing the child's exposure to lead.

Children with symptomatic lead poisoning, with and without encephalopathy, should be managed by a multidisciplinary team.

Asymptomatic children with blood lead levels  $\geq 45 \mu\text{g/dL}$  should receive chelation therapy.

Different clinical centers and programs use different protocols to medically manage children with blood lead levels of 25 to  $44 \mu\text{g/dL}$ .

**The single most important factor in managing of childhood lead poisoning is reducing the child's exposure to lead; some children, however, will benefit from chelation therapy.** One approach for pharmacologic treatment of children with lead poisoning follows. It is a general guide and is not the only pharmacologic regimen that can be used to treat poisoned children.

### **Medical Management of Symptomatic Lead Poisoning (with or without Encephalopathy)**

**General Management.** Children with symptomatic lead poisoning (with or without encephalopathy) must be treated only at a pediatric center that has an intensive care unit. They should be managed by a multidisciplinary team that includes, as needed, critical care, toxicology, neurology, and neurosurgery. The child's neurological status and fluid balance must be carefully monitored.

The symptoms associated with lead poisoning (with or without lead encephalopathy) are described on page 51. One or more of those symptoms associated with an elevated blood lead level constitutes an acute medical emergency. Because chelation regimens are the same for cases of symptomatic lead poisoning (with and without encephalopathy), guidelines for clinical management have been included in a single section.

**Chelation therapy.** Although succimer has been approved for chelation of children with blood lead levels  $>45 \mu\text{g/dL}$ , experience in treating symptomatic children is limited. Therefore, the treatment regimen discussed here uses  $\text{CaNa}_2\text{EDTA}$  and BAL. Chelation with succimer is discussed in more detail on page 57.

Start treatment with a dose of 75 mg/m<sup>2</sup> BAL only, given by deep intramuscular injection; administer BAL at a dose of 450 mg/m<sup>2</sup>/day in divided doses of 75 mg/m<sup>2</sup> every 4 hours. Once this dose is given and an adequate urine flow is established, administer CaNa<sub>2</sub>EDTA at a dose of 1,500 mg/m<sup>2</sup>/day. Give CaNa<sub>2</sub>EDTA as a continuous intravenous infusion in dextrose and water or in a 0.9% saline solution. The concentration of CaNa<sub>2</sub>EDTA should not exceed 0.5% in the parenteral fluid. (When treating a child with encephalopathy, the physician may choose to give CaNa<sub>2</sub>EDTA intramuscularly to reduce the amount of fluid administered.) Treat with combined BAL-CaNa<sub>2</sub>EDTA therapy for a total of 5 days. During treatment, monitor renal and hepatic function and serum electrolyte levels daily (Piomelli et al., 1984).

A second course of chelation therapy with CaNa<sub>2</sub>EDTA alone (at blood lead levels 45-69 µg/dL) or combined with BAL (at blood lead levels 70 µg/dL), may be required once there is a rebound in the blood lead level after chelation. Wait at least 2 days before giving a second course of chelation. A third course is required only if the blood lead concentration rebounds to a value >45 µg/dL within 48 hours after the second course of treatment. Unless there are unusual and compelling clinical reasons, wait at least 5 to 7 days before beginning a third course of CaNa<sub>2</sub>EDTA (Piomelli et al., 1984).

### **Medical Management of Asymptomatic Lead Poisoning**

Clinical management of asymptomatic lead-poisoned children with blood lead levels high enough to require chelation is similar to that of symptomatic children. Focus on reducing the child's exposure to lead and decreasing the child's body burden of lead.

Although succimer has been approved for chelation of children with blood lead levels >45 µg/dL, experience with this drug is limited. Therefore, the treatment regimen discussed here uses CaNa<sub>2</sub>EDTA and BAL. Chelation with succimer is discussed in more detail on page 57.

**Blood lead level ≥70 µg/dL.** Children with blood lead levels ≥70 µg/dL (with or without symptoms) represent an acute medical emergency. If the blood lead level is ≥70 µg/dL, give both BAL and CaNa<sub>2</sub>EDTA in the same doses and using the guidelines as for treatment of symptomatic lead poisoning (Page 59). A second course of chelation therapy with CaNa<sub>2</sub>EDTA alone may be required if the blood lead concentration rebounds to a value ≥45 µg/dL within 5 to 7 days after treatment. In general allow at least 5 to 7 days before beginning a second course of CaNa<sub>2</sub>EDTA. Some practitioners give a second course of chelation after a 3-day rest period if the immediate post-treatment blood lead level is >35µg/dL (J. Chisolm, personal communication).

**Blood lead level 45 to 69 µg/dL.** If the blood lead value is between 45 and 69 µg/dL, chelation treatment should be limited to CaNa<sub>2</sub>EDTA only. CaNa<sub>2</sub>EDTA is given for 5 days at a dose of 1,000 mg/m<sup>2</sup>/day intravenously by continuous infusion or in divided doses, as described on page 56. During treatment, evaluate renal and hepatic function and serum electrolyte levels regularly. Do not continue CaNa<sub>2</sub>EDTA treatment for more than 5 days (Piomelli et al., 1984).

A second course of chelation therapy with CaNa<sub>2</sub>EDTA alone may be required if the blood lead level rebounds to 45 µg/dL within 7 to 14 days after treatment. Allow 5 to 7 days before beginning a second course of CaNa<sub>2</sub>EDTA.

**Blood lead level 25 to 44 µg/dL.** For this blood lead range, the effectiveness of chelation therapy in decreasing the adverse effects of lead on children's intelligence has not been shown. Treatment regimens vary from clinic to clinic. Some practitioners treat children with lead levels

in this range pharmacologically. (Although it is not approved for this use, some use D-penicillamine for children in this blood lead range.) The minimum medical management for children with these blood lead levels is to decrease the children's exposure to all sources of lead, to correct any iron deficiency and maintain an adequate calcium intake, and to test frequently to ensure that the child's blood lead levels are decreasing. Many experienced practitioners decide whether to use chelation therapy on the basis of the results of carefully performed  $\text{CaNa}_2\text{EDTA}$  mobilization tests (Page 53).

**Blood lead level 20 to 24  $\mu\text{g}/\text{dL}$ . Only very minimal data exists about chelating children with blood lead levels below 25  $\mu\text{g}/\text{dL}$ , and such children should not be chelated except in the context of approved clinical trials.** A child with a confirmed blood lead level of 20 to 24  $\mu\text{g}/\text{dL}$  will require individual case management by a pediatric health-care provider. The child should have an evaluation with special attention to nutritional and iron status. The parents should be taught about 1) the causes and effects of lead poisoning, 2) the need for more routine blood lead testing, 3) possible sources of lead intake and how to reduce them, 4) the importance of adequate nutrition and of foods high in iron and calcium, and 5) resources for further information. (This is described in more detail in Chapter 4.) Sequential measurements of blood lead levels along with review of the child's clinical status should be done at least every 3 months. Iron deficiency should be treated promptly. Children with blood lead levels in this range should be referred for environmental investigation and management. **Identifying and eradicating all sources of excessive lead exposure is the most important intervention for decreasing blood lead levels (Chapter 8).**

## POST-CHELATION FOLLOWUP

Recheck blood lead levels 7 to 21 days after treatment. Determine if retreatment is necessary.

Do not discharge a child from the hospital until a lead free environment can be assured.

At the end of each treatment cycle, the blood lead concentration usually declines to  $<25 \mu\text{g}/\text{dL}$ . Within a few days, however, reequilibration among body lead compartments takes place and may result in a rebound; thus, **the blood lead level must be rechecked 7 to 21 days after treatment to determine whether retreatment is necessary** (Piomelli et al., 1984; Chisolm et al., 1985).

Children who undergo chelation treatment require long-term followup preferably from pediatric health-care providers, nutritionists, environmental specialists, and community outreach workers. Community outreach workers provide a critical bridge between hospital-based or clinic-based (outpatient) medical care, health advocacy education, and environmental remediation outside the hospital. Children should **never** be discharged from the hospital **until they can go to a lead-free environment** (CDC, 1985; Piomelli et al., 1984). Lead-free safe housing (with friends, relatives, or in designated transitional housing), in which a treated child can live during the entire abatement process through the post-abatement clean-up, must be arranged. With appropriately carried-out public health measures, complete and safe abatement should be achieved during the treatment period (CDC, 1985).

Once a child is discharged to a safe environment, frequent followup is mandatory. In general, depending on the initial blood lead value, most children who require chelation therapy must be followed closely for at least one year or more. All children undergoing chelation treatment should be seen every other week for 6-8 weeks, then once a month for 4-6 months. A child treated with BAL and  $\text{CaNa}_2\text{EDTA}$  should be followed more closely: weekly for 4 to 6 weeks, then monthly for 12 months.

At each clinic visit, housing information should be updated. If history suggests that exposure is increasing or if blood lead levels are rising, the dwelling must be reinspected to evaluate the possibility of new sources of environmental lead, inadequate abatement, or unsound structures in buildings (for example, poor plumbing with leaks) that cause further chipping or breakdown of a previously repaired dwelling (Piomelli et al., 1984).

## **RESEARCH AREAS AND FUTURE TRENDS IN THE MANAGEMENT OF CHILDHOOD LEAD POISONING**

### **Further evaluation is needed on**

Xray fluorescence (XRF) measurements of lead in bone.

Efficacy of chelating agents in reducing the adverse neurobehavioral effects of lead.

Uses of succimer.

Toxicity of  $\text{CaNa}_2\text{EDTA}$  and other chelating agents.

### **Bone Lead Measurements Using Xray Fluorescence (XRF)**

According to published data, L-line and the K-line XRF techniques permit non-invasive assessment of skeletal lead stores. These bone stores reflect the lead burden accumulated over an individual's life. In contrast, blood lead values reflect recent lead exposure and absorption during the past 1 to 3 months and provide limited information about lead toxicokinetics over time (Rabinowitz et al., 1977). Evaluations using the L-line methodology in children have shown that blood lead levels underestimate the body burden of lead in lead-poisoned children (Rosen et al., in press); and sequential measurements of lead in lead-poisoned children by the L-line technique have shown decreases in bone lead after  $\text{CaNa}_2\text{EDTA}$  treatment or environmental intervention (Rosen et al., in press). K-line techniques have been used mainly to measure bone lead levels in workers. Quantitation of bone lead content of children takes about 16 minutes.

At present, XRF equipment is available only in a few centers in the United States and Europe.

### **Efficacy of Chelating Agents**

The benefits of chelation therapy in symptomatic lead-poisoned children are well known (Chisolm, 1968). Prompt intervention with chelating agents prevents progression to symptomatic disease and normalizes biochemical indices of lead toxicity. However, the efficacy of chelating agents in reversing or modifying the adverse neurobehavioral effects at all blood lead

levels in apparently asymptomatic children needs to be carefully assessed. Better understanding of this issue is critical in deciding the end-point of medical treatment. It is also essential in defining when chelation should be used.

### **Succimer**

Data are needed on the tissue sites of lead chelated by succimer, the adverse effects of succimer, the effect of succimer on absorption of lead from the gastrointestinal tract, and the effectiveness of different dose regimens of succimer. Assuming that no new significant adverse effects are noted after succimer is used more widely, the efficacy and appropriate use of succimer for treating lead-poisoned children with blood lead levels below 45 µg/dL needs to be established.

### **Toxicity of CaNa<sub>2</sub>EDTA**

Results of one animal study suggest that CaNa<sub>2</sub>EDTA may transiently increase brain lead levels (Cory-Slechta et al., 1987). The redistribution of lead during chelation needs further study.

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### **References**

Aposhian HV, Aposhian MM. Meso-2,3-dimercaptosuccinic acid: chemical, pharmacological and toxicological properties of an orally effective metal chelating agent. *Ann Rev Pharmacol Toxicol* 1990; 30:279-306.

CDC (Centers for Disease Control). 1985. Preventing lead poisoning in young children: A statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

Chisolm JJ Jr. The use of chelating agents in the treatment of acute and chronic lead intoxication in childhood. *J Pediatr* 1968;73:1-38.

Chisolm JJ Jr. Management of increased lead absorption - illustrative cases. In: Chisolm JJ Jr, O'Hara DM, editors. *Lead absorption in children: management, clinical, environmental aspects*. Baltimore: Urban and Schwarzenberg, 1982:171-88.

Chisolm JJ Jr, Mellits ED, Quaskey SA. The relationship between the level of lead absorption in children and the age, type, and condition of housing. *Environ Res* 1985;38:31-45.

Chisolm JJ Jr. Evaluation of the potential role of chelating therapy in the treatment of low to moderate lead exposures. *Environ Health Perspect* 1990;89:67-74.

Cory-Slechta DA, Weiss B, Cox C. Mobilization and redistribution of lead over the course of calcium disodium ethylenediamine tetraacetate chelation therapy. *J Pharmacol Exp Ther* 1987;243:804-13.

Graziano JH, LoIacono NJ, Meyer P. Dose-response study of oral 2,3-dimercaptosuccinic acid in children with elevated blood lead concentrations. *J Pediatr* 1988;113:751-7.

Kapoor SC, Wielopolski L, Graziano JH, LoIacono NJ. Influence of 2,3-dimercaptosuccinic acid on gastrointestinal lead absorption and whole-body lead retention. *Toxic Appl Pharmacol* 1989;97:525-9.

Markowitz ME, Rosen JF. Assessment of lead stores in children: validation of an 8-hour  $\text{CaNa}_2\text{EDTA}$  provocative test. *J Pediatr* 1984;104:337-2.

Markowitz ME, Rosen JF, Bijur PE. Effects of iron deficiency on lead excretion in children with moderate lead intoxication. *J Pediatr* 1990;116:360-4.

Markowitz ME, Rosen JF. Need for the lead mobilization test in children with lead poisoning. *J Pediatr* 1991;119:305-10.

Osterloh J, Becker CE. Pharmacokinetics of  $\text{CaNa}_2\text{EDTA}$  and chelation of lead in renal failure. *Clin Pharm Ther* 1986;40:686-93.

Perlstein MA, Attala R. Neurologic sequelae of plumbism in children. *Clin Pediatr* 1966;5:292-8.

Piomelli S, Corash L, Corash MB, Seaman C, Mushak P, Glover B, Padgett R. Blood lead concentrations in a remote Himalayan population. *Science* 1980;210:1135-7.

Piomelli S, Rosen JF, Chisolm JJ Jr, Graef JW. Management of childhood lead poisoning. *J Pediatr* 1984;105:523-32.

Rabinowitz MB, Wetherill GW, Kopple JD. Magnitude of lead intake from respiration by normal man. *J Lab Clin Med* 1977;90:238-48.

Rosen JF, Markowitz ME, Bijur PE, Jenks ST, Wielopolski L, Kalef-Ezra JA, Slatkin DN. L-line x-ray fluorescence of cortical bone lead compared with the  $\text{CaNa}_2\text{EDTA}$ -treated lead-toxic children. *Environ Health Perspect* (in press).

Shannon M, Graef J, Lovejoy FH Jr. Efficacy and toxicity of D-penicillamine in low-level lead poisoning. *J Pediatr* 1988;112:799-804.

Weinberger HL, Post EM, Schneider T, Helu B, Friedman J. An analysis of 248 initial mobilization tests performed on an ambulatory basis. *Am J Dis Child* 1987;141:1266-70.

## Chapter 8. Management of Lead Hazards In The Environment of The Individual Child

### *Summary*

*To eradicate childhood lead poisoning, lead hazards must be abated.*

*Environmental case management includes a number of actions prescribed for a child with lead poisoning.*

*Precautions must be taken to ensure that abatement is conducted in the safest and most effective manner possible.*

Eradicating childhood lead poisoning requires a long-term active program of primary lead-poisoning prevention, including abatement of lead-based paint hazards in homes, day-care centers, and other places where young children play and live. For the child who is lead poisoned, however, efficient and effective interventions are needed as quickly as possible. Abatement means making the source of lead inaccessible to the child.

Lead-based paint is the most common source of high-dose lead poisoning. Complete abatement of lead-based paint means eliminating all lead-based paint in a housing unit as a source of lead for the child, either by removing the paint or by using permanent barriers. Complete abatement of the lead hazards in the child's environment is the most effective and only certain way to prevent further damage. Complete abatement is expensive, but once a dwelling is abated, many generations of children may live in that home and reap the benefits. Unfortunately, complete abatement may not always be possible, and shorter term, preventive maintenance procedures may have to be undertaken to minimize the potential for further damage.

Lead-based paint is rarely completely abated in many of the largest childhood lead poisoning prevention programs. Instead, various degrees of incomplete abatement—designed to eliminate the worst hazards and prevent near-term exposures—are conducted. Development of cost-effective, safe, simple, and widely applicable methods of complete paint abatement is a high priority.

Whether complete abatement or preventive maintenance is done, persons performing the work should be knowledgeable of the hazards of lead to themselves, to children, and to the environment. They should be trained in the proper procedures for abatement and preventive maintenance, since improperly performed work can actually increase the hazards to the child.

Each situation in which a child gets poisoned is unique and must be evaluated by a person or team of persons skilled and knowledgeable about lead poisoning, hazard identification, and interventions to reduce lead exposure, including abatement of lead-based paint in housing. Childhood lead poisoning prevention programs need to work closely with other relevant agencies (for example, housing and environmental agencies) to ensure that the quickest and most effective approach is taken to remediating the environments of poisoned children.

The 1985 CDC statement on *Preventing Lead Poisoning in Young Children* set the level for environmental intervention at 25 µg/dL. In this new statement CDC recommends environmental intervention for children with blood lead levels of  $\geq 20$  µg/dL, or of  $\geq 15$  µg/dL that persist. Where resources are limited, however, individual environmental intervention must first focus on those children with the highest blood lead levels. CDC also recommends that environmental interventions be directed at primary prevention of lead poisoning in communities with a large number or percentage of children with blood lead levels  $\geq 10$  µg/dL (Chapter 9).

When resources are limited, environmental intervention must first focus on those children with the highest blood lead levels. When possible, abatement should be conducted for primary prevention of lead poisoning.

The Department of Housing and Urban Development has issued *Lead-Based Paint Interim Guidelines for Hazard Identification and Abatement in Public and Indian Housing*, hereafter called the HUD Guidelines (HUD, 1990, also published in the Federal Register 55FR14556). (The worker protection guidance was subsequently revised and published in the Federal Register, 55FR39873.) This document is referenced frequently in this chapter because it contains the most comprehensive information on identifying and abating lead-based paint hazards available. It is not expected that every childhood lead poisoning prevention program or every homeowner will follow the guidelines completely. These guidelines were written for lead hazards in public and Indian housing, particularly for use during comprehensive modernization programs. Such programs, carried out when the property is vacant and in multiple units at one time, offer opportunities for very thorough and complete abatements. Most abatement of lead-based paint in the private sector does not occur in such a context. In the private sector, abatement is generally done in occupied housing scattered throughout an area, often with limited resources. In the context of this chapter, the HUD guidelines are an information source on identifying and abating hazards.

## ENVIRONMENTAL CASE MANAGEMENT

### **Environmental case management includes**

- Educating parents about the sources, effects, and prevention of lead poisoning.
- Investigating the environment to identify lead sources and effectively communicating the results of this investigation.
- Taking emergency measures to reduce lead exposure.
- Doing long-term interventions to reduce lead exposure.
- Evaluating the efficacy of the interventions.

Environmental case management includes a number of actions prescribed for a child with lead poisoning. Ideally, environmental case management should be conducted by a team of professionals in public health, environmental activities, medical management, and social

management. A team approach to intervention will help ensure that followup is timely and effective. The management team may need to solve many related problems, such as whether to investigate supplemental addresses, where to find temporary alternative housing, and how to use community resources to assist the family in dealing with the lead-poisoned child.

A team approach to case management is most effective when all team members:

1. Demonstrate professionalism.
2. Show genuine concern for the poisoned child and family.
3. Support other team members.
4. Use similar terms, descriptions, and reference points to communicate with the child's family.
5. Meet specific time frames for followup.
6. Reinforce education of the family at every encounter.

### **Time Frames for Investigations and Interventions**

The following guidelines describe the maximum time within which environmental interventions should be implemented. All children with blood lead levels  $\geq 20$   $\mu\text{g/dL}$  should have environmental interventions conducted as quickly as possible. Children with blood lead levels  $\geq 45$   $\mu\text{g/dL}$  require prompt chelation therapy. **The homes of these children must be remediated before they are allowed to return.**

**Blood lead levels  $\geq 70$   $\mu\text{g/dL}$ .** Children with blood lead levels above 69  $\mu\text{g/dL}$  constitute a medical emergency and must be hospitalized immediately. They are at highest risk for severe, permanent neurologic damage due to lead exposure and must be given highest priority for followup. Environmental investigation and intervention should be started within 24-48 hours and should include the child's home and potential sites of exposure, such as a relative's home or a day-care center. The homes of these children must be remediated before they are allowed to return.

**Blood lead levels between 45 and 69  $\mu\text{g/dL}$ .** These children can be given a slightly lower intervention priority than the children classified as medical emergencies. Environmental investigation and intervention should begin within 5 working days and should include the same components as for children with higher blood lead levels. The homes of these children must be remediated before they are allowed to return.

**Blood lead levels between 20 and 44  $\mu\text{g/dL}$ .** Environmental investigation and intervention should begin within 10 working days. Since many of these children will not be hospitalized and since allowing exposures to continue might lead to further increases in blood lead levels, environmental interventions for these children should be conducted as quickly as possible.

**Blood lead levels between 15 and 19  $\mu\text{g/dL}$ .** Environmental investigation and intervention for children at this level should be based upon program resources and the ability of program staff to respond. At a minimum, these children and their families should have education regarding lead poisoning. If blood lead levels  $\geq 15$   $\mu\text{g/dL}$  persist, environmental intervention should be made where possible—including assisting the parents in locating potential sources of lead contamination in and around the home and instructing them about how to reduce the risk of lead contamination. If resources permit, a full environmental inspection for lead-based paint should be done for such children.

Although full environmental investigation and abatement is not recommended as part of the management of children with blood lead levels below 15 µg/dL, the identification and reduction of lead hazards in all high-risk housing is an important primary prevention measure (Chapter 9).

### **Educating Parents about Lead Poisoning**

The parents of all lead-poisoned children should be educated about lead poisoning. In communities with a high incidence of lead poisoning, communitywide educational efforts should be considered. These efforts should provide information similar to that in the anticipatory guidance provided by pediatric health care providers. Information provided should include:

1. Causes and effects of lead poisoning.
2. Relationship of the child's blood lead level to the potential for adverse health effects.
3. Need for followup blood lead testing of the child.
4. The child's possible sources of lead intake and practical means for reducing and eliminating these sources.
5. Role of nutrition in decreasing lead absorption.
6. Resources where parents can get further information (addresses and telephone numbers of local health-care providers or public health agencies).

Ideally, this information should be provided during a face-to-face meeting with the parents. When local resources are limited, however, written material (in an appropriate language) may be mailed to the child's family. Educating parents about lead poisoning is further discussed in Chapter 4.

### **Investigating the Environment and Communicating the Results**

The technical aspects of inspecting a home for lead-based paint are discussed below. In general, an investigation of the environment of a lead poisoned child should include the following steps:

1. Determine the most likely sources of high-dose exposure to lead.
2. Investigate the child's home to identify possible sources of lead. Include both the interior and exterior environment and give special attention to painted surfaces, dust, soil, and water. (Details on how to test for lead-based paint are in the next section.)
3. Advise parents and caretakers about identified and potential sources of lead and ways to reduce exposure.
4. In cases in which the parent does not own the home, notify the property owner immediately that a child residing on the property has lead poisoning. Discuss the results of the environmental investigation and the abatement interventions required with the property owner. Emphasize the importance of prompt abatement. When a child with a medical emergency from lead poisoning is identified, an immediate, face-to-face meeting with the property owner may best demonstrate the need for emergency intervention.
5. Advise parents and property owners that no residents or personal belongings should remain in the home during abatement.

6. Monitor the effectiveness and timeliness of abatement procedures closely.
7. Coordinate environmental activities with those of other professionals, including the health-care providers and persons responsible for public health and social management. A team approach to intervention will help provide a timely and effective followup.

### **Emergency Measures to Reduce Lead Exposure**

The first phase of environmental intervention may be to use short-term emergency interventions to **temporarily** reduce lead hazards. As soon as a blood lead level  $\geq 20$   $\mu\text{g/dL}$  (or, if resources permit,  $\geq 15$   $\mu\text{g/dL}$ ) is confirmed, parents should be advised of the hazards of lead-based paint and lead dust. They should be told not to attempt abatement themselves—improper abatement will most likely **increase** lead dust levels in the home and create additional, more severe exposure for the child. The temporary nature of interventions other than abatement should be emphasized.

When the source of lead is paint and paint-contaminated dust, parents can be instructed to stabilize the paint, wet-mop all floors, and wet-clean window sills and window wells at least twice per week. Cleaners high in phosphates appear to work particularly well. Sponges and rags used in this cleaning should be used for no other purpose. In particular, they should not be used to wash dishes or clean eating- or food-preparation surfaces, since dangerous contamination could result. Children's hands should be washed regularly, particularly before eating. Toys and pacifiers that are mouthed should be washed at least daily. Cribs and playpens should be moved away from chipping or peeling paint; furniture can be placed in front of areas that are not intact to make them less accessible. Dry sweeping of dust should be avoided, because it will stir up and spread the dust. Other measures to reduce lead exposure are discussed in Chapter 4.

### **Long-Term Measures to Reduce Lead Exposure**

The next phase of environmental intervention involves long-term hazard reduction. If the source of lead is paint and paint-contaminated dust, the lead hazards are permanently abated only when all lead-based paint is completely removed or otherwise made permanently inaccessible. Less extensive practices, which are commonly used by childhood lead poisoning prevention programs, may be called "long term abatement." Certain maintenance procedures (for example, frequent cleaning and keeping walls freshly painted) may be classified as "preventive maintenance," but in general these procedures offer no absolute assurance of safety. In cases other than "permanent abatement," how long the hazard will remain under control depends on such factors as the quality of the workmanship, the thoroughness of the procedure, the soundness of the underlying structure, and the condition of the plumbing and roof. Moisture from leaky pipes or roofs can quickly cause paint that was smooth and intact to blister and scale, generating hazardous levels of lead dust. Except in unusual situations (such as in the case of housing that is not likely to be viable for more than a couple of years or when no alternative housing is available), temporary measures to reduce exposure should not be a substitute for abatement or an excuse for delaying abatement.

Technical aspects of lead-based paint abatement are discussed below.

## Evaluating Intervention Activities

The effectiveness of any intervention for a lead-poisoned child should be evaluated by its impact on the child's blood lead level. Measurement of environmental lead levels may also be helpful.

## Assessing the Lead Problem in the Child's Community

If a number of children are identified as being lead-poisoned in a community, communitywide interventions as described in Chapter 9 should be considered.

## TESTING FOR AND ABATING LEAD-BASED PAINT

Tests for measuring the lead content of paint on walls have limitations; new tests for evaluating lead in paint are being developed.

Proper abatement must be done by experts; untrained parents, property owners, workers or contractors should not attempt it.

**NOTE: Remodeling or repainting homes with lead-based paint should be considered just as hazardous as abatement. Whenever lead-based paint must be disturbed by sanding, scraping, heating, or other forms of abrasion, the same precautions should be taken for remodeling or repainting as for abatement itself.**

## Inspection and Testing

Several methods are available for determining the lead content of paint. These include XRF, wet chemical methods, and chemical spot tests. Although XRF analyzers are convenient, instruments available at the present time have limitations. A study by the National Institute of Standards and Technology (NIST, 1989) indicated possible substrate errors in the direct-reading XRF's of as much as  $\pm 2$  mg/cm<sup>2</sup>. These errors were caused by differences in base materials in walls and trim. (At very high readings, for example, above 3 mg/cm<sup>2</sup>, this error has no practical significance). The spectrum analyzer, while considerably more expensive than the direct reader, provided much more accurate results. Only fully trained and experienced personnel should use XRF analyzers.

Wet chemical methods of analysis must be used if an XRF machine is not available or if it produces ambiguous results. Wet chemical methods require that a paint chip sample with all layers of paint on the surface be sent to a laboratory for analysis. Wet chemical analysis has two major disadvantages—results are not available immediately, and it is expensive.

Like XRF, chemical spot tests are performed on-site. A scratch is made through all layers of paint, and a chemical is placed on the scratch. If the scratch turns certain colors, further evaluation is needed. Chemical spot tests are qualitative, not quantitative, and the interpretation of the results is subjective. These tests are being refined and evaluated as to their safety, accuracy, and reliability.

Further information on proper testing procedures for lead-based paint is in the NIST study report and the HUD Guidelines.

The 1985 CDC statement on lead recommended an XRF value of 0.7 mg/cm<sup>2</sup> as the maximum level of lead in paint in a residence. The HUD standard, mandated by Congress, is 1.0 mg/cm<sup>2</sup>. Several states have established their own XRF standards for lead in paint; these standards range from 0.7 mg/cm<sup>2</sup> to 1.2 mg/cm<sup>2</sup>. The HUD document and some state regulations use a standard of 0.5% lead by weight for laboratory analysis.

Lead in paint should always be considered a “potential” hazard. An **immediate** lead hazard exists when lead-based paint is 1) chipping, peeling or flaking; 2) is chalking, thereby producing lead dust; 3) is on a part of a window which is abraded through the opening and closing of the window; 4) is on any surface which is walked on (like floors) or otherwise abraded; 5) can be mouthed by a child (for example, window sills); or 6) is disturbed by repainting or remodeling. A potential lead hazard can easily become an immediate hazard through natural aging, plumbing or roof leaks, or the paint being disturbed. All lead-based paint exceeding the action level should, therefore, be abated whenever possible. Otherwise, complicated records must be kept of unabated surfaces, and those surfaces must be inspected frequently to make certain that they have not become immediate hazards.

When inspecting for lead-based paint hazards, care must be taken to evaluate **all** types of surfaces, including walls, ceilings, doors and windows, trim and jambs, woodwork, stairway components, porch components, garages, sheds, fences, play equipment, and any other structures on the premises. Because of legal requirements in some areas, it may be necessary to test every surface that may be painted with lead paint (that is, every window, every door, every piece of trim, etc.). Often, however, abatement decisions can be made without this costly and time-consuming approach. Even with an XRF, a full inspection of all surfaces in an average home may take 4 hours or more. Sometimes, extrapolating XRF results to untested surfaces may make sense. Such extrapolation, however, should only be used for positive results. For example, if test results for one window are positive for lead, it is safe to assume that all similar windows are painted with lead-based paint; if test results for one window are negative, it is **not** safe to assume that no windows have lead-based paint.

Recent studies have indicated that many children are poisoned by lead-contaminated dust ingested through normal hand-to-mouth activity. This dust can come from lead contaminated soil that is tracked into the home on shoes or from the clothes of a parent who works with lead. However, the most common source of lead dust in the average old house is lead-based paint. Some believe that the level of lead dust in a house can be used as a measure of the severity of the immediate hazard.

## **Abatement**

Proper abatement includes the following steps:

1. Proper training of all workers involved in the abatement.
2. Protecting those workers whenever they are in the abatement area.
3. Containing lead-bearing dust and debris.
4. Replacing, encapsulating, or removing lead-based paint.
5. Cleaning the abatement area thoroughly.
6. Disposing of abatement debris properly.
7. Inspecting to make certain the property is ready for reoccupancy.

Abatement should never be attempted by untrained parents, property owners, or contractors. The property owner's responsibility is not met until all the above steps have been completed.

**Preparation:** Just prior to abatement, all personal belongings, movable furniture, and drapes should be removed from the abatement area. In homes with deteriorated lead-based paint, furniture may be highly contaminated with lead dust. It is recommended that badly soiled carpets and drapes be discarded because of the difficulty of removing lead from them. Furniture should be cleaned before it is returned to the abated dwelling or it should be replaced. Wood, metal, glass and plastic surfaces should be washed with a high phosphate detergent. If possible, all upholstered furniture, carpets, drapes, and bare surfaces should be vacuumed with a High Efficiency Particle Accumulator (HEPA).

**Precautions:** Residents and their belongings should remain out of their homes during abatement. Under no circumstances should children and pregnant women be allowed to enter the dwelling unit during the abatement because abatement can generate large quantities of hazardous lead dust.

**Training:** All workers involved in a lead abatement project should be properly trained in the following: health effects of lead; proper procedures for worker protection, including procedures for personal hygiene and for wearing and caring for respirators; containment of an abatement project; various methods for abating lead-based paint and the safety and environmental hazards involved with each; and procedures for transporting and disposing of abatement debris properly.

**Worker Protection:** All workers on a lead abatement project and their families must be protected from the hazardous lead dust that will be generated. The minimum acceptable protection would be coveralls (preferably disposable); shoe coverings; hair covering; gloves; goggles; and a properly fitted, negative-pressure, half-mask respirator with a HEPA filter. Other, more protective respirators may be needed to protect from hazards such as organic vapors. If the abatement methods used would generate significant quantities of lead dust or organic vapors, workers must wear more protective respirators, such as supplied air-respirators.

The potential hazard to workers of lead dust **ingestion** is as significant, if not more significant, than inhalation. Workers must not eat, drink, or smoke on the job; and hands and face must be washed before breaks and at the end of the day. On-site showers should, if possible, be provided. If on-site showers are not available, workers must shower and wash their hair immediately upon returning home. They must be careful not to carry hazardous levels of lead dust home on their bodies, shoes, or clothing. Therefore, work clothes should not be worn home; either workers should wear protective workclothes instead of street clothes at the worksite or they should wear protective garments over their street clothes. Work clothes should be disposed of or laundered by the employer to prevent the contamination of automobiles, homes, etc. with dust; lead-contaminated clothing should be handled with care and should not be laundered with other clothing of the worker or his family.

*Note: The chapter in the HUD guidelines on worker protection was revised and published separately in the Federal Register on September 28, 1990 (55FR39873).*

**Containment:** The work area should be contained with plastic (6 mil) to protect other living areas, yards, heating and ventilation systems, etc. from contamination. All nonmovable furnishings, such as counters, cabinets, and radiators should be covered with plastic. All floors should also be covered with plastic to prevent lead dust from being deposited in cracks and crevices and from being ground into the surface during the abatement.

**Abatement:** Abatement methods fall into three categories: 1) replacement, 2) encapsulation or enclosure, and 3) paint removal. These categories are discussed in more detail as follows:

**Replacement:** Removing the building component (such as a window, door, or baseboard) and replacing it with a new one.

**Encapsulation:** Covering a lead-painted surface with a material that will effectively prevent access to the lead-based paint and that will also prevent lead-bearing dust from that surface from entering the living environment.

**Paint Removal:** Stripping paint by heat, chemical, or mechanical means. This can be done either on-site or at the premises of a chemical stripping firm.

Certain methods of removing lead-based paint may be particularly hazardous to both the worker and the building occupants and may be banned in some areas. They are—

1. Removing paint with an open-flame torch or other heating device that operates at temperatures likely to volatilize lead (the melting point of lead is 621 °F).
2. Machine sanding surfaces with lead-based paint.
3. Sand blasting lead-based paint, except when the equipment is fitted with a vacuum device that prevents the dispersal of the debris.
4. Uncontained hydro-blasting.
5. Using chemical strippers containing methylene chloride. Methylene chloride is extremely toxic and protecting workers from exposure to this chemical is difficult.

If possible, all surfaces painted with lead-based paint should be abated by replacement, encapsulation, or paint removal. Ordinary paint is never an appropriate encapsulant; it is only part of a temporary maintenance procedure. Encapsulation materials should be durable and, where possible, affixed with both fasteners and adhesive. Paintlike coatings should be used with caution. Only coatings and adhesives that are proven to be safe and effective should be used. Any material that will eventually chip, peel, or flake upon aging or from water damage is not appropriate.

Paint removal is potentially the most hazardous abatement method because considerable amounts of lead dust and lead residue are generated. Paint removal from porous surfaces, such as wood or concrete, **always** leaves significant amounts of lead residue. This residue may not be visible and removing it requires extremely vigorous cleaning procedures (alternating washing with a high phosphate detergent and HEPA vacuuming (see below)). Painting over this residue can lead to lead dust problems when this paint begins to deteriorate or when it is abraded. Of particular concern are friction surfaces, such as window and door jambs.

Workers using any method that generates large volumes of dust or fumes should use caution. Such methods increase the difficulty of worker protection and the likelihood that hazardous levels of lead-bearing dust will remain in the dwelling unit or be deposited in the soil surrounding the home. Demolishing older structures with lead-based paint likewise can result in deposition of lead-bearing dust into the soil or on neighboring property, and dust suppression techniques should be used.

**Clean-Up:** All lead abatement activity is likely to generate quantities of hazardous lead dust. Unless this dust is properly cleaned, the dwelling unit will be more hazardous after abatement than it was before. This dust is difficult to remove. Daily clean-up, consisting of misting debris with water, carefully sweeping it, and placing it in double 4-mil or 6-mil plastic bags, is necessary to minimize the risk to workers of accumulated lead dust.

After abatement and before repainting, all surfaces in the dwelling must be thoroughly vacuumed with a HEPA vacuum; wet washed, preferably with a high phosphate detergent such as tri-sodium phosphate; and then vacuumed again. The property should be visually inspected before being repainted. The inspector should ascertain that all surfaces covered with lead-based paint have been abated and that no visible dust or debris remains on site.

Several states have adopted a post-abatement dust standard which has been included in the HUD Guidelines. This standard was set mainly on the basis of practicality rather than a health or risk assessment, and further research is needed on the adequacy and appropriateness of that standard. The standard allows the following maximum levels of lead in dust:

Floors	200 $\mu\text{g}/\text{ft}^2$
Window Sills	500 $\mu\text{g}/\text{ft}^2$
Window Wells	800 $\mu\text{g}/\text{ft}^2$

Inspectors and persons collecting dust samples and laboratories measuring dust lead levels should be thoroughly familiar with the recommended sampling and analysis protocols for dust in the HUD Guidelines.

After the inspection, abated surfaces should be repainted, if appropriate. Wooden floors should receive a coat of deck enamel or urethane, concrete floors should be sealed with deck enamel, and linoleum or tile floors should be waxed. Sealing the floors will bind any remaining dust particles and enable the occupants to clean those surfaces easily.

**Disposal:** Certain wastes from a lead-based paint abatement project, either liquid or solid, may be classified as hazardous. If so, they will have to be treated as such and handled by a licensed transporter or treatment firm. In any case, **all** debris from an abatement project, whether classified as hazardous or not, must be contained and transported in such a way as to prevent the dispersal of lead bearing dust, chips, or liquid into the environment. Lead debris should never be sent to a solid waste incinerator, a disposal method that disperses lead into the air.

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### **References**

HUD (Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

NIST (National Institute for Standards and Technology). Methods for measuring lead concentrations in paint films. Washington (DC): NIST, 1989.

## Chapter 9. Management of Lead Hazards In The Community

### *Community Level Intervention Includes*

*Screening and surveillance.*

*Risk assessment and integrated prevention planning.*

*Outreach and education.*

*Infrastructure development.*

*Hazard reduction.*

In theory, primary prevention has always been the goal of childhood lead poisoning prevention programs. In practice, however, most programs focus exclusively on secondary prevention, dealing with children who have already been poisoned. As programs shift the emphasis to primary prevention, their efforts must be designed to systematically identify and remediate environmental sources of lead, including, most importantly, dwellings containing old lead paint.

The shift from case management to community-level intervention will require a fundamental shift in perspective. The focus must shift from the individual child to the population of children at risk and the environment in which they live. The purpose of community-level intervention is to identify and respond to sources, not cases, of lead poisoning. The responsibility for addressing lead poisoning will have to be expanded beyond health agencies to include a variety of housing, environmental, and social service agencies at the local, county, state, and national level.

To be successful, community-level intervention will involve at least five types of activities:

1. **Screening and surveillance:** Determining populations at risk and the locations of the worst exposures.
2. **Risk assessment and integrated prevention planning:** Analyzing all available data to assess sources of lead, exposure patterns, and high-risk populations and developing primary prevention plans.
3. **Outreach and education:** Informing health-care providers, parents, property owners, and other key people about lead poisoning prevention.
4. **Infrastructure development:** Finding the resources needed for a successful program of risk reduction.
5. **Hazard reduction:** Reducing the hazards of lead-based paint and lead in dust and soil, particularly in high-risk buildings and neighborhoods.

## **SURVEILLANCE**

### **To identify the highest risks**

Collect data on blood lead levels.  
Conduct environmental surveys.  
Collect demographic data.

For the most effective allocation of resources, data on the extent of the lead poisoning problems and the location of the worst lead hazards must be available for study. By combining data on blood lead levels, environmental sources of lead, and community demographics, public health agencies can identify and quantify the risk of lead poisoning in the community.

### **Data on Blood Lead Levels**

Results of regular blood lead screening for pre-school children (as recommended in Chapter 6 of this report) will eventually provide an important source of information on the distribution of lead hazards in a community. Current data, which are based on limited public screening or the experience of practitioners or clinics, cannot provide the true prevalence of elevated blood lead levels in the children of a community. Communities may need to undertake additional, focused screening surveys to obtain data on the prevalence of elevated blood lead levels. Even after near-universal screening is in place, such targeted screening efforts will continue to be necessary in areas and populations in which substantial numbers of children do not have regular pediatric health-care providers. To be accurate, such surveys should use door-to-door (rather than fixed-site) sampling and blood lead (rather than EP) analysis.

Health officials can evaluate risks better if they have the results of all blood lead tests, not just the elevated blood lead levels. A convenient mechanism for gathering such information is for laboratories to report all blood lead testing results to an appropriate local or state health agency. Where mandatory reporting is not in place, health agencies should work with laboratories and pediatric health-care providers to obtain as much data as possible on blood lead test results.

### **Environmental Surveys**

Environmental surveys that are designed to identify the common sources of childhood lead exposure can be undertaken in conjunction with or as a complement to community-based surveys of blood lead levels. Environmental surveys do not, however, replace measurement of children's blood lead levels. The environmental sources and pathways of lead that can be assessed in environmental surveys include lead-based paint, lead in dust and soil, lead in drinking water, lead from industrial sources and wastes, and lead from unusual sources such as folk medicines or ceramicware.

An environmental survey of the sources of lead around children's homes (paint, dust, and soil) can be undertaken in conjunction with a door-to-door blood lead screening program. A team would consist of a nurse or phlebotomist who would obtain the blood samples and an inspector who could use the most cost-effective combination of measurements of lead in dust,

soil, and paint (for example, XRF analyzers, chemical spot tests, or removal of paint chips for laboratory analysis). When screening for lead-based paint in housing, inspectors should obtain representative data on the prevalence of hazards and need not undertake the type of comprehensive inspections described in Chapter 8. Protocols for environmental sampling must be developed, and inspectors must be trained in sampling techniques before the survey program begins.

In addition to looking for lead hazards in housing, a comprehensive environmental lead testing program could look for other lead sources, including drinking water in schools and residential buildings, soil in playgrounds and schoolyards, street dusts, and lead-based paint in nonresidential buildings such as day-care centers and schools. In some cases, environmental data obtained for other purposes may be useful. For example, the federal Safe Drinking Water Act and Lead Contamination Control Act requires some testing for lead in drinking water, so health officials could, therefore, contact water suppliers and school officials to obtain test results. Agricultural extension services may have data on lead levels in soil.

### **Demographic Data**

Health surveys, such as the National Health and Nutrition Examination Survey (NHANES), have correlated children's blood lead levels with demographic factors such as family income and place of residence (for example, center city vs. suburbs). Demographic data now becoming available from the 1990 census can be used to broadly identify high-risk areas. Variables to consider include the age of housing (pre-1960 housing has the most lead), income levels, socioeconomic status, ethnicity, and the number or density of preschool children in the area. For best results, communities would use this demographic information to predict where the greatest lead hazards might be located and then to conduct appropriate blood lead or environmental surveys to see if the predictions are true. Once the most predictive demographic variables have been identified, algorithms or survey instruments could be designed to accurately predict which areas pose the greatest risk on the basis of demographic data alone.

## **RISK ASSESSMENT AND INTEGRATED PREVENTION PLANNING**

Risk assessment involves using all available data to evaluate community lead hazards.

Primary prevention planning should include representatives from the private and public sectors.

A primary prevention plan should include outreach and education programs, infrastructure development, and hazard reduction.

Public health officials should use all of the information at their disposal—blood lead screening results, environmental survey data, and demographic information—to create the most accurate picture of community lead hazards, including sources of lead, exposure patterns, and high-risk populations. Whenever possible, officials should focus on specific sources and the smallest pertinent geographic area of concern. In some new suburban communities, for example, the risks may not justify a communitywide program to abate lead-based paint in

housing. Nevertheless, there may be a need to address specific sources (for example, drinking water in new houses with lead solder) or specific neighborhoods (for example, an old part of town where Victorian homes are being rehabilitated).

Because lead poisoning is completely preventable, public health officials should assess the success of current prevention efforts. Local communities should focus on how well the hazards of lead are being addressed in that community, rather than on whether the community has a bigger or smaller lead problem than other communities.

Once a decision is made to address at least some aspects of the lead problem in a community, public health officials should develop an integrated primary prevention plan. The plan should be assembled with input from other agencies (including housing and environmental agencies), pediatric health-care providers, parents, teachers, community groups, and other interested persons. The plan should identify which sources, geographic areas, or high-risk populations are to be addressed. Each element of the plan should include a description of who will have the primary responsibility for implementation, where financial and other resources will be obtained, and a time schedule for implementation. Plans should be as specific as possible in order to allow public officials and community groups to periodically assess whether and how the plan is being carried out.

The remaining sections of this chapter address in more detail three types of activities that should be addressed in any comprehensive primary prevention plan: outreach and education, infrastructure development, and hazard abatement.

## **OUTREACH AND EDUCATION**

Must take place during every phase of the community activity.

Should involve many agencies and both the public and private sectors.

Should involve many people in various professions, including those related to real estate.

Outreach and education must take place during every phase of the community activity, beginning before health and environmental screening and ending when risk abatement is complete. Among the most important targets for outreach and educational programs are local officials, health-care providers, parents, property owners, day-care providers, and early childhood educators. The outreach programs can be carried out through pamphlets and other written materials, local news media, public meetings, school programs, and social service agencies.

Local health officials who have traditionally carried out all or most lead poisoning prevention activities in a community must begin by reaching out to other agencies that will have a role in communitywide primary prevention efforts. When possible, lead poisoning prevention should be part of an integrated program for creating safe and affordable housing or for providing poor people in the community with the full range of needed social services. Local, state, and federal agencies dealing with health, housing, environmental, and children's issues should be contacted.

Many health-care providers are unaware of the most recent developments in the field of lead poisoning prevention. Educational campaigns by local officials, licensing agencies, professional

associations, clinics, and hospitals are needed to ensure that pediatric health-care providers understand current thinking about the health and environmental aspects of lead poisoning. Outreach through pamphlets, grand rounds, and continuing education programs should be targeted to pediatricians, family practitioners, pediatric and community health nurses, obstetricians, and midwives.

For parents, including pregnant women, initial education should focus on the hazards of lead and the need for blood lead testing of children at regular intervals. Parents should know about risk factors that warrant frequent screening (Page 42). Educational materials should help parents understand the implications of the screening results. Finally, parents (and parents-to-be) should be informed about simple steps that can be taken to reduce risks, such as proper nutrition and housekeeping measures (Chapter 4). Such outreach efforts can be targeted to individual parents and to groups of parents and prospective parents.

Property owners and managers, realtors, and other real estate professionals need to learn how to maintain property in a safe and habitable condition. Banks, mortgage companies, and insurance companies could play an important role in conveying this information at critical junctures, such as when a property owner is buying a property or seeking financing for major renovations. In addition, property owners should be given written material that explains how to remove lead safely.

Day-care providers and early childhood educators should be given information about lead poisoning and its sequelae. Those taking care of young children should also be informed about the need to identify and abate lead hazards in day-care buildings and schools. Parents of lead-poisoned children can aid in this process by informing their child's teachers about the past lead poisoning, so that the teacher can make better informed decisions about the need for remedial measures.

## **INFRASTRUCTURE DEVELOPMENT**

### **Infrastructure development includes**

- Regulations and rules on removing lead.
- Trained inspection and abatement contractors.
- Temporary housing for families whose homes are undergoing abatement.
- Financial resources for lead poisoning prevention activities, including abatement.

Before a community can launch a broad-based program of preventive deleading and hazard reduction, many elements must be in place to support such activities.

First, regulations or other rules and standards are needed to define when and how inspections and deleading are to occur. One local agency (housing, environmental, or health) should be designated as the lead agency with respect to community intervention activities and a system should be put in place for coordinating regulatory and other activities among all involved agencies.

A second requirement is contractors who are trained 1) to identify lead hazards, including lead-based paint, and 2) to remove lead-based paint safely. Besides inspectors, abatement planners, contractors, supervisors, and workers are needed. Optimally, such persons should be licensed or certified by a federal or state agency to ensure that their work is of high quality.

A third infrastructure need is temporary housing for families during the deleading process. Because lead-based paint should not be removed while homes and apartments are occupied, communities must develop strategies to provide temporary alternative housing for families that need it. Communities should consider developing “safe houses” where families can live temporarily at little or no cost while their homes are being delead. If families are encouraged to “double up” with friends, measures should be in place to ensure that the home or apartment being used for temporary housing is free of lead hazards.

The final element of infrastructure involves financial resources for both the government agencies overseeing lead poisoning prevention programs and property owners or tenants seeking to delead. This may be the most difficult element, yet it is critical to a successful program. Existing federal and state housing funds (for example, Community Development Block Grants) can be used to finance lead removal if communities so choose. Starting in Fiscal Year 1992, a limited number of loans for abatement may be available from the Department of Housing and Urban Development through the HOME program.

## **HAZARD ABATEMENT**

Hazard abatement may involve a number of activities directed at multiple environmental sources and pathways.

Abatement resources should be targeted to the highest risk neighborhoods and homes.

The goal of hazard abatement is the systematic elimination of lead hazards in the community.

The final and most important step is actually abating the lead hazards. This may involve many activities, such as corrosion control to reduce the amount of lead in drinking water and covering or removing lead-contaminated soil in parks and playgrounds. In many cases, the primary risk will be lead-based paint and the primary form of risk reduction will be preventive deleading—abatement that occurs before children have been poisoned. Before the hazard abatement phase, the community must decide which lead hazards to target. Information gathered during risk assessment should be used to ensure that abatement resources are directed toward the highest risk neighborhoods and buildings.

Local officials have a variety of means at their disposal to promote preventive deleading—from education and outreach, programs designed to increase voluntary deleading, financial assistance to encourage deleading, and regulatory mechanisms to require deleading. If voluntary efforts are to be encouraged, outreach must go beyond general information to provide building owners with specific information about how to survey a building for lead hazards and how to abate those hazards.

If abatement is mandated by law, the law should require safe and effective abatements. Rental property owners should not be permitted to avoid abating their properties by evicting or refusing to rent to families with young children.

Whatever mechanisms are used, the goal of hazard abatement must be to systematically eradicate the lead hazards in the community. Such a program will protect not only lead-poisoned children but all children—and thus safeguard the community’s future.

# **Appendix I.**

## **Capillary Sampling Protocol**

Microspecimens of blood collected by fingerstick are widely used to measure lead levels, yet there is no consensus on what constitutes the best collection procedure. Published data on collection methods are scant, and much of the data that do exist were published 10 or more years ago, when technology was not as advanced and blood lead levels of concern were significantly higher.

The high potential for lead contamination of capillary specimens during collection is well known (CDC, 1985; DeSilva and Donnan, 1980; Mitchell et al., 1974), and the special steps used to minimize the likelihood of contamination constitute the major differences among collection procedures. Special procedures used for minimizing contamination include thorough scrubbing of the hand and finger with soap and then alcohol (Sinclair and Dohnt, 1984; NECCLPP, 1985); using dilute nitric acid (Rosen, 1972; MHD, 1988); or using silicone or a similar barrier spray (Lyngbye et al., 1990; CDHS, 1990; NYSDH, 1989; Mitchell et al., 1974).

Several types of containers for collecting children's blood (maximum volume  $\leq 500 \mu\text{L}$ ) have been introduced in recent years and are widely used by screening programs. The new containers are better than glass tubes, since glass capillary tubes are very fragile. Whether these new containers are suitable for collecting blood for lead measurement has not been extensively studied.

More research on these and other issues is clearly needed before the best fingerstick collection procedures can be identified. Recognizing these constraints, a fingerstick procedure for collecting blood lead specimens follows.

### **A. NEEDED MATERIALS**

1. Soap.
2. Alcohol swabs. If a surgical or other disinfectant soap is used, alcohol swabs can be eliminated.
3. Sterile cotton balls or gauze pads.
4. Silicone spray or swabs. The benefits of using a barrier spray, which forms a layer between the skin and blood droplets, have been debated. In addition to doubts about the spray's effectiveness in reducing specimen contamination, the spray makes the collection more expensive and complex. Some evidence exists, however, the spray reduces contamination (NYSDH, 1989; Mitchell et al., 1974), so it is included in this procedure.
5. Examination gloves.
6. Lancets. The type of lancet used is largely a matter of personal preference, so long as sterility is guaranteed.
7. Collection containers. If glass capillary tubes are used, sealing clay or tube caps will also be required. No additional supplies are needed for most other microcontainers. The laboratory should be consulted to ensure that an appropriate size capillary tube is used.
8. Adhesive bandages.

9. Trash bags suitable for medical waste and containers for sharps. Bags containing medical waste should be clearly identified as such.
10. Storage or mailing containers if needed. If specimens require shipment, follow the Postal Service or other appropriate regulations for shipping body fluids.

Materials used in the collection procedure that could contaminate the specimen (for example, blood containers, alcohol swabs, and barrier sprays) must be lead-free. **Before selecting equipment for use in blood collection, consult with the laboratory about its requirements.** In many cases, the laboratory will recommend or supply suitable collection equipment and may precheck the equipment for lead contamination. Some instrument manufacturers also supply collection materials that are pretested for lead content.

## **B. PREPARING FOR BLOOD COLLECTION**

All personnel who collect specimens should be well-trained in and thoroughly familiar with the collection procedure. The skill of the collector will greatly influence the specimen quality. All equipment should be within easy reach. The environment should be clean, secure, and as nonthreatening to the child as possible. Any necessary consent should be obtained before specimen collection begins, and the procedure should be explained to the child and the parent or guardian. Used materials should be discarded into appropriate waste containers suitable for medical waste immediately following use.

## **C. PREPARING THE FINGER FOR PUNCTURE**

**NOTE: Puncturing of the fingers of infants less than 1 year of age is not recommended. Puncturing of the heel is more suitable for these children (NCCLS, 1986).**

Collection personnel should wear examination gloves whenever the potential for contact with blood exists. If the gloves are coated with powder, it should be rinsed off with tap water.

The child's hands should be thoroughly washed with soap and then dried with a clean, low lint towel. If water is unavailable, foam soaps can be used without water (D. Griffin, Louisville/Jefferson County Department of Health, personal communication). Plain, unprinted, nonrecycled towels are best (WSLH, 1985). If desired, a brush can be used for cleaning the finger; brushing during washing can increase blood circulation in the finger (CDHS, 1990). Once washed, the finger must not be allowed to come into contact with any surface, including the child's other fingers.

The finger to be punctured (often the middle finger) must be free of any visible infection or wound; it should be massaged to increase circulation before being punctured with the lancet. This can be accomplished during or after washing (NYSDH, 1989; CDHS, 1990).

### **Steps for Preparing the Child's Finger**

1. Select examination gloves. If necessary, rinse them to remove powder.
2. Wash the child's hands thoroughly with soap and water, and then dry them with an appropriate towel.
3. Grasp the finger that has been selected for puncture between your thumb and index finger with the palm of the child's hand facing up.

4. If not done during washing (see preceding notes), massage the fleshy portion of the finger gently.
5. Clean the ball or pad of the finger to be punctured with the alcohol swab. Dry the fingertip using the sterile gauze or cotton ball.
6. Apply the silicone barrier. If a spray is used, shake the can vigorously to mix the contents. Direct the spray away from child and collector. Silicone does not dry, and the finger can be punctured immediately.

#### **D. PUNCTURING OF THE FINGER AND FORMING DROPS OF BLOOD**

1. Grasp the finger and quickly puncture it with a sterile lancet in a position slightly lateral of the center of the fingertip.
2. Wipe off the first droplet of blood with the sterile gauze or cotton ball.
3. If blood flow is inadequate, gently message the proximal portion of the finger and then press firmly on the distal joint of the finger. A well-beaded drop of blood should form at the puncture site.
4. Do not let the blood run down the finger or onto the fingernail.

After the finger is ready, the puncture and subsequent steps of forming a drop of blood and filling the collection container should be performed quickly and efficiently, since any delay can make collection more difficult (for example, the blood may clot or the child may resist). Several types of lancets are suitable for puncturing children's fingers. The range from small manual lancet blades to spring-loaded assemblies. Regardless of the lancet used, the puncture should be made swiftly and cleanly and should be deep enough to allow adequate flow.

The site of the puncture should be slightly lateral to the ball of the finger. This region is generally less calloused, which makes puncturing easier and, possibly less painful (CDHS, 1990). The first drop of blood contains tissue fluids that will produce inaccurate results; it should be removed with a sterile gauze or cotton ball (NYSDH, 1989; CDHS, 1990).

A barrier material such as silicone will help a distinct "bead" of blood to form, which aids collection. Blood that runs down the finger or around the fingernail is no longer suitable. Blood flows better if the punctured finger is kept lower than the heart. Inadequate blood flow can be improved by gently massaging the proximal portion of the finger in a distal direction, then pressing firmly at the distal joint of the punctured finger (restricting blood flow out of the fingertip) and *gently* squeezing the sides of the fingertip. Excessive squeezing will cause tissue fluid to be expressed, and the fluid will compromise specimen integrity (NYSDH, 1989; CDHS, 1990). Do not let the blood run down the finger or fingernail.

## E. FILLING THE COLLECTION CONTAINER

1. Continuing to grasp the finger, touch the tip of the collection container to the beaded drop of blood.
2. Draw the blood into the container maintaining continuous flow of blood.
3. When full, cap or seal the container as appropriate.
4. Agitate the specimen to mix the anticoagulant through the blood.
5. Check that the container is properly labeled, and place it in an appropriate storage area.
6. Stop the bleeding and cover the finger with an adhesive bandage. Bleeding should stop very quickly. If bleeding is slow to stop, apply pressure to the puncture site with a sterile gauze or a cotton ball. If bleeding continues after 3 to 5 minutes of applying pressure, consult a physician.

The proper procedure for filling and capping collection containers is somewhat specific to the container used. As a general rule, contact between the skin and the container is to be avoided. To prevent clotting of the specimen, blood must be mixed with the anticoagulant after filling the container. Depending on the container and anticoagulant used, the agitation needed can range from gentle rocking to vigorous shaking. Some procedures call for the collection container to be rotated during filling so that anticoagulant will be distributed quickly through the sample (MDPH, 1990).

To facilitate blood flow, many procedures call for the collection container be held nearly horizontal, with a slight downward angle. Blood flow into the container should be uninterrupted to avoid air bubbles in the specimen. Except for glass capillary tubes, containers come with appropriate caps, and these should be applied immediately following collection. Specimens in glass capillary tubes are often collected in duplicate and then sealed with rubber caps or plasticine sealing clay or both. Again, consulting with the laboratory and knowing the manufacturer's recommendations are important to ensure specimen integrity and suitability for analysis.

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### **References**

CDHS (California Department of Health Services). Childhood blood lead screening: fingerstick blood sampling method. Berkeley (CA): CDHS, 1990.

CDC (Centers for Disease Control). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

De Silva PE, Donnan MB. Blood lead levels in Victorian Children. *Med J Aust* 1980;1:93.

Lynghye T, Jorgensen PJ, Grandjean P, Hansen ON. Validity and interpretation of blood lead levels: a study of Danish school children. *Scand J Clin Lab Invest* 1990;50:441-9.

MDPH (Massachusetts Department of Public Health). Procedure for obtaining fingerstick blood samples. Jamaica Plain (MA): MDPH, Childhood Lead Poisoning Program, 1990.

MHD (Milwaukee Health Department). Generalized procedure fingerstick blood (hematocrit and/or lead test). Milwaukee: MHD, 1988.

Mitchell DG, Aldous KM, Ryan FJ. Mass screening for lead poisoning: capillary blood sampling and automated Delves-cup atomic-absorption analysis. *N Y State J Med* 1974;74:1599-603.

NCCLS (National Committee for Clinical Laboratory Standards). Procedures for the collection of diagnostic blood specimens by skin puncture—second edition. NCCLS publication H4-A2. Villanova (PA): NCCLS, 1986.

NECCLP (New England Consortium of Childhood Lead Poisoning Programs). New England public health laboratory lead testing services [report]. Providence (RI): NECCLPP, 1985.

NYSDH (New York State Department of Health). Blood lead and erythrocyte protoporphyrin: a recommended procedure for collecting fingerstick blood specimens. Albany (NY): NYSDH, Wadsworth Center for Laboratories and Research, 1989.

Rosen JF. The microdetermination of blood lead in children by flameless atomic absorption: the carbon rod atomizer. *J Lab Clin Med* 1972;80:567-76.

Sinclair DF, Dohnt BR. Sampling and analysis techniques used in a blood lead survey of 1241 children in Port Pirie, South Australia. *Clin Chem* 1984;10:1616-9.

WSLH (Wisconsin State Laboratory of Hygiene). Complete sampling instructions for capillary lead. Madison (WI): WSLH, University of Wisconsin Center for Health Sciences, 1985.

## Appendix II. Summary For The Pediatric Health-Care Provider

The following material summarizes those parts of the lead statement that are most important for the pediatric health-care provider. It does not include some of the critical information on such topics as primary prevention, sources of lead in the environment, and abatement. More information on all of the topics described herein is included in the complete statement.

### CHAPTERS 1 AND 2. INTRODUCTION AND BACKGROUND

Childhood lead poisoning is one of the most common pediatric health problems in the United States today, and it is entirely preventable. Enough is now known about the sources and pathways of lead exposure and about ways of preventing this exposure to begin the efforts to permanently eradicate this disease. The persistence of lead poisoning in the United States, in light of all that is known, presents a singular and direct challenge to public health authorities, clinicians, regulatory agencies, and society.

Previous lead statements issued by the Centers for Disease Control (CDC) have acknowledged the adverse effects of lead at lower and lower levels. In the most recent previous CDC lead statement, published in 1985, the threshold for action was set at a blood lead level of 25  $\mu\text{g}/\text{dL}$ , although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that some adverse effects occur at blood lead levels at least as low as 10  $\mu\text{g}/\text{dL}$  in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure.

Because 10  $\mu\text{g}/\text{dL}$  is the lower level of the range at which effects are now identified, primary prevention activities—communitywide environmental interventions and nutritional and educational campaigns—should be directed at reducing children's blood lead levels at least to below 10  $\mu\text{g}/\text{dL}$ . Blood lead levels between 10 and 14  $\mu\text{g}/\text{dL}$  are in a border zone. While the overall goal is to reduce children's blood lead levels below 10  $\mu\text{g}/\text{dL}$ , there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14  $\mu\text{g}/\text{dL}$ . First, laboratory measurements of blood lead levels may be variable, so a blood lead level in this range may, in fact, be below 10  $\mu\text{g}/\text{dL}$ . Secondly, effective environmental and medical interventions for children with blood lead levels in this range have not yet been identified and evaluated. Finally, the sheer numbers of children in this range would preclude effective case management and would detract from the individualized followup required by children who have higher blood lead levels.

**The single, all-purpose definition of childhood lead poisoning has been replaced with a multitier approach.** Community prevention activities should be triggered by blood lead levels  $\geq 10$   $\mu\text{g}/\text{dL}$ . Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels  $\geq 20$   $\mu\text{g}/\text{dL}$ . All children with blood lead levels  $\geq 15$   $\mu\text{g}/\text{dL}$  require individual followup, including nutritional and educational interventions. Furthermore, depending on the availability of resources environmental investigation and remediation should be done for children with blood lead levels of 15-19  $\mu\text{g}/\text{dL}$ , if such levels persist. The highest priority should continue to be the children with the highest blood lead levels.

Other differences between the 1985 and 1991 statements are as follows:

**Screening test of choice.** Because the erythrocyte protoporphyrin level is not sensitive enough to identify children with elevated blood lead levels below about 25  $\mu\text{g}/\text{dL}$ , the screening test of choice is now blood lead measurement.

**Universal screening.** Since virtually all children are at risk for lead poisoning, a phase in of universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement.

**Primary prevention.** Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require communitywide environmental interventions, as well as educational and nutritional campaigns.

**Succimer.** In January, 1991, the U.S. Food and Drug Administration approved succimer, an oral chelating agent, for chelation of children with blood lead levels over 45  $\mu\text{g}/\text{dL}$ .

### CHAPTER 3. SOURCES AND PATHWAYS OF LEAD EXPOSURE

A child's environment is full of lead. Children are exposed to lead from different sources (such as paint, gasoline, and solder) and through different pathways (such as air, food, water, dust, and soil). Although all U.S. children are exposed to some lead from food, air, dust, and soil, some children are exposed to high dose sources of lead. Lead-based paint is the most widespread and dangerous high-dose source of lead exposure for preschool children.

Lead-based paint (containing up to 50% lead) was in widespread use through the 1940s. Although the use and manufacture of interior lead-based paint declined during the 1950s and thereafter, exterior lead-based paint and lesser amounts of interior lead-based paint continued to be available until the mid-1970s. (Lead-based paint produced after the 1940s tended to have much lower lead concentrations than lead-based paint produced earlier.)

Pica, the repeated ingestion of nonfood substances, has been implicated in cases of lead poisoning; however, a child does not have to eat paint chips to become poisoned. More commonly, children ingest dust and soil contaminated with lead from paint which flaked or chalked as it aged or which has been disturbed during home maintenance and renovation. This lead-contaminated house dust, ingested via normal repetitive hand-to-mouth activity, is now recognized as a major contributor to the total body burden of lead in children. Because of the critical role of dust as an exposure pathway, children living in sub-standard housing and in homes undergoing renovation are at particular risk for lead poisoning.

Many cases of childhood lead poisoning that result from renovation or remodelling of homes have been reported. Before older homes undergo any renovation that may generate dust, they should be tested for the presence of lead-based paint. If such paint is found, contractors experienced in working with lead-based paint should do the renovations.

Other potentially important sources and pathways of lead exposure include soil and dust, water, "take home" exposures from parental occupations and hobbies, water, and food. Very high-dose exposure may occasionally result from sources other than lead-based paint in specific situations.

## CHAPTER 4. THE ROLE OF THE PEDIATRIC HEALTH-CARE PROVIDER

Pediatric health-care providers, working as part of the public health team, must play a critical role in the prevention and management of childhood lead poisoning. Their roles include 1) educating parents about key causes of childhood lead poisoning; 2) screening children and interpreting blood lead test results; 3) working with appropriate groups in the public and private sectors to make sure that poisoned children receive appropriate medical, environmental, and social service followup; and 4) coordinating with public health officials and others involved in lead-poisoning prevention activities.

Along with educating parents about nutrition and developmental stages, providers should discuss the potential hazards of lead. They should focus on the major likely preventable sources of high-dose lead poisoning in their communities. Parents should be told of the potential dangers of peeling lead-based paint, the potential hazards of renovating older homes, and the need for good work practices if their occupations or hobbies expose them to lead. In some communities parents should be warned about the potential for lead exposure from improperly fired ceramicware and imported pottery. In others, where water lead levels are a concern, parents could be advised to use only fully-flushed water (that is, water that has not been standing in pipes for a prolonged time) from the cold-water tap for drinking, cooking, or preparing infant formula.

Pediatric health-care providers should provide information about simple ways parents can reduce exposure to lead. Some examples of these are discussed below.

**Housekeeping Interventions.** Particularly in older homes, which may have been painted with lead-based paint, interventions to reduce exposure to dust may help reduce blood lead levels. These include:

- Make sure your child does not have access to peeling paint. Pay special attention to windows and window sills and wells.
- If the house was built before about 1960 and has hard surface floors, wet mop them at least once a week with a high phosphate solution (for example, 5-8% phosphates). (The phosphate content of automatic dishwashing detergents and other cleaning substances is often listed on the label and may be high enough for this purpose. Otherwise, trisodium phosphate can be purchased in hardware stores.) Other hard surfaces (such as window sills and baseboards) should also be wiped with a similar solution. Do not vacuum hard surface floors or window sills or wells, since this will disperse dust. Vacuum cleaners with agitators remove dust from rugs more effectively than vacuum cleaners with suction only.
- Wash your child's hands and face before he/she eats.
- Wash toys and pacifiers frequently.

### **Other Interventions to Reduce Exposure to Lead.**

- If soil around the home is or is likely to be contaminated with lead (for example, if the home was built before 1960 or the house is near a major highway), plant grass or other ground cover. Since the highest concentrations of lead in a yard tend to be near surfaces that were once painted with lead paint, like exterior walls, if exterior lead paint was likely to be used, plant bushes around the outside of your house so your child cannot play there.

- In areas where the lead content of water exceeds the drinking water standard, use only fully-flushed water from the cold-water tap for drinking, cooking, and making formula. In communities where water conservation is a concern, use first-flush water for other purposes.
- Do not store food in open cans, particularly if the cans are imported.
- Do not use pottery or ceramicware that was improperly fired or is meant for decorative use for food storage or service.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies (Chapter 3).

Not all aspects of a poisoned child's followup will be managed by the pediatric health-care provider, although the provider is an important part of the team. Through his or her interactions with the child and family and the responsible public health agency, the provider should make sure that any appropriate interventions are occurring. If the child needs a medical evaluation (for a blood lead level  $\geq 20$   $\mu\text{g/dL}$ ) or pharmacologic treatment (Chapter 7), either the provider should do it or should refer the child to a place that treats large numbers of poisoned children. The provider should make sure that the child receives an appropriate environmental investigation and remediation with the help of the public health agencies. Particularly if the child is developmentally delayed, the provider should refer the child to an appropriate infant stimulation or child development program. In many cases, lead-poisoned children and their families will also benefit from social services followup.

## CHAPTER 5. THE ROLE OF STATE AND LOCAL PUBLIC AGENCIES

A variety of local, state and federal agencies play a role in preventing childhood lead poisoning. Pediatric health care providers and parents should know about what these agencies do so that they can use these resources effectively. In turn, these agencies must coordinate their activities to ensure that all aspects of childhood lead poisoning prevention—health, housing, and environment—are being addressed, and to provide the most comprehensive and cost-effective services to at-risk children, their parents, and their health-care providers.

## CHAPTER 6. SCREENING

Traditionally, the main purpose of a childhood lead poisoning screening program has been to identify asymptomatic lead-poisoned children and to intervene as quickly as possible to reduce their blood lead levels. An additional benefit of screening programs is that abatement of lead sources for poisoned children results in prevention of lead poisoning for children who would have been exposed to those sources in the future. As the focus in lead poisoning prevention turns more to primary prevention, an additional benefit of screening is that data generated can be used in targeting interventions to places with children at high risk for lead poisoning.

In 1984, the last year for which estimates are available, it is believed that between 3 and 4 million children younger than age 6 years (17% of all U.S. children in this age group) had blood lead levels above 15  $\mu\text{g/dL}$ . Furthermore, about 74% of occupied, privately owned housing built before 1980 contains lead-based paint (defined as  $\geq 1$   $\text{mg/cm}^2$ ). **Because almost all U.S.**

**children are at risk for lead poisoning (although some children are at higher risk than others), our goal is that all children should be screened, unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem.** (Deciding that no problem exists requires that a large number or percentage of children be tested.) The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement. Children at highest risk for lead poisoning are the highest priority for screening. Table 6-1 provides guidance on the groups for which repeated screening is most strongly indicated.

Children ages 6 to 72 months who live in or are frequent visitors to deteriorated old buildings, including day care centers, make up the highest priority group. Because the highest concentrations of lead in paint were used in the early 1900s, homes built before about 1960 are of greatest concern. Children whose homes are being renovated are also at extremely high risk. Since siblings, housemates, visitors, and playmates of children with confirmed lead poisoning may have similar exposures to lead, they also should be promptly screened. In communities with a high prevalence of lead poisoning, health departments should consider door-to-door screening, since many children with lead poisoning may be missed by fixed-site screening.

Children with parents whose work or hobbies involve lead may also risk lead exposure (Chapter 3). Also, children living near lead smelters or other industries where lead is processed may be at increased risk for lead poisoning.

In general, screening and assessment for lead poisoning should focus on children younger than 72 months of age, particularly on children younger than 36 months of age. Young children engage in the most hand-to-mouth activity (and therefore are at highest risk for lead exposure) and have the most rapidly developing nervous systems, making them more vulnerable to the effects of lead. Children with developmental delays, who may exhibit pica or have more extensive hand-to-mouth activity than other children, would be expected to be at increased risk for lead poisoning even if they are 72 months of age and older. These children may have to be screened more often during early infancy, and may require screening into their school years.

Children who have unexplained seizures, neurological symptoms, abdominal pain, or other symptoms that are consistent with lead poisoning should also have their blood lead levels measured. In addition, the possibility of lead poisoning should be considered in any child with growth failure, developmental delay, hyperactivity, behavior disorders, hearing loss, anemia, etc.

**Table 6-1. Priority groups for screening**

- 
- Children, ages 6 to 72 months, who live in or are frequent visitors to deteriorated housing built before 1960.
  - Children, ages 6 to 72 months, who live in housing built before 1960 with recent, ongoing, or planned renovation or remodeling.
  - Children, ages 6 to 72 months, who are siblings, housemates, or playmates of children with known lead poisoning.
  - Children, ages 6 to 72 months, whose parents or other household members participate in a lead-related occupation or hobby.
  - Children, ages 6 to 72 months, who live near active lead smelters, battery recycling plants, or other industries likely to result in atmospheric lead release.
-

## Screening Method

Since erythrocyte protoporphyrin (EP) is not sensitive enough to identify more than a small percentage of children with blood lead levels between 10 and 25  $\mu\text{g}/\text{dL}$  and misses many children with blood lead levels  $\geq 25 \mu\text{g}/\text{dL}$ , measurement of blood lead levels should replace the EP test as the primary screening method. Unless contamination of capillary blood samples can be prevented, lead levels should be measured on venous samples. Obtaining capillary specimens is more feasible at many screening sites. Contamination of capillary specimens obtained by finger prick can be minimized if trained personnel follow proper technique. Elevated blood lead results obtained on capillary specimens should be considered presumptive and must be confirmed using venous blood. At the present time, not all laboratories will measure lead levels on capillary specimens.

## Anticipatory Guidance and Assessing Risk

Guidance on childhood lead poisoning prevention and assessment of the risk of lead poisoning should be part of routine pediatric care. Anticipatory guidance is discussed in detail in Chapter 4. The guidance and risk assessment should emphasize the sources and exposures that are of greatest concern in the child's community (Chapter 3). Because lead-based paint has been used in housing throughout the United States, in most communities it will be necessary to focus on this source.

Table 6-2 has sample questions for assessing a child's risk for high-dose lead exposure. Starting at 6 months of age and at each regular office visit thereafter, pediatric health-care providers should discuss childhood lead poisoning and assess the child's risk for high-dose exposure. The questions asked should be tailored to the likely sources of exposure in the community. **The questions are not a substitute for a blood lead test.**

On the basis of responses to questions such as those in Table 6-2, children can be categorized as low or high risk for high-dose lead exposure. If the answers to all questions are negative, the child is at low risk for high-dose lead exposure and should be screened by a blood lead test at 12 months and again, if possible, at 24 months (since blood lead levels often peak at ages greater than 12 months). If the answer to any question is positive, the child is potentially at high risk for high-dose lead exposure, and a blood lead test should be obtained. **For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.**

**Table 6-2. Assessing the risk of high-dose exposure to lead—sample questionnaire**

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*Does your child—*

1. Live in or regularly visit a house with peeling or chipping paint built before 1960? This could include a day care center, preschool, the home of a babysitter or a relative, etc.
  2. Live in or regularly visit a house built before 1960 with recent, ongoing, or planned renovation or remodeling?
  3. Have a brother or sister, housemate, or playmate being followed or treated for lead poisoning (that is, blood lead  $\geq 15 \mu\text{g}/\text{dL}$ )?
  4. Live with an adult whose job or hobby involves exposure to lead (see Chapter 3)?
  5. Live near an active lead smelter, battery recycling plant, or other industry likely to release lead?
-

## Screening Schedule

The following sections provide a minimum screening schedule for children aged 6 up to 36 and 36 to 72 months. The schedule is not rigid. Rather, it is a guide for pediatric health-care providers and screening programs to use in conjunction with other pertinent information in determining when an individual child should be tested. Programs and pediatric health-care providers may choose to screen more frequently than described below.

### ***Children 6 up to 36 months of age:***

A questionnaire should be used at each routine office visit to assess the potential for high-dose lead exposure and, therefore, the appropriate frequency of screening.

#### ***Schedule if the child is at low risk for high dose lead exposure by questionnaire:***

A child at *low risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 12 months of age.

If the 12-month blood lead result is  $<10 \mu\text{g/dL}$ , the child should be retested at 24 months if possible, since that is when blood lead levels peak.

If a blood lead test result is  $10\text{-}14 \mu\text{g/dL}$ , the child should be retested every 3 to 4 months. After 2 consecutive measurements are  $<10 \mu\text{g/dL}$  or three are  $<15 \mu\text{g/dL}$ , the child should be retested in a year.

If any blood lead test result is  $\geq 15 \mu\text{g/dL}$ , the child needs individual case management and should be retested at least every 3 to 4 months (Page 96).

#### ***Schedule if the child is at high risk for high dose lead exposure by questionnaire:***

A child at *high risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 6 months of age.

If the initial blood lead result is  $<10 \mu\text{g/dL}$ , the child should be rescreened every 6 months. After 2 subsequent consecutive measurements are  $<10 \mu\text{g/dL}$  or three are  $<15 \mu\text{g/dL}$ , testing frequency can be decreased to once a year.

If a blood lead test result is  $10\text{-}14 \mu\text{g/dL}$ , the child should be screened every 3 to 4 months. Once 2 subsequent consecutive measurements are  $<10 \mu\text{g/dL}$  or three are  $<15 \mu\text{g/dL}$ , testing frequency can be decreased to once a year.

If any blood lead test result is  $\geq 15 \mu\text{g/dL}$ , the child needs individual case management and should be retested at least every 3 to 4 months (Page 94).

### ***Children $\geq 36$ months and $< 72$ months of age:***

As for younger children, a questionnaire should be used at each routine office visit of children from 36 to 72 months of age. Any child at high risk by questionnaire who has not previously had a blood lead test should be tested. All children who have had venous blood lead tests  $\geq 15 \mu\text{g/dL}$  or who are at high risk by questionnaire should be screened at least once a year until their sixth birthday (age 72 months) or later, if indicated (for example, a retarded child with pica). Children should also be rescreened any time history suggests exposure has increased. Children with blood lead levels  $\geq 15 \mu\text{g/dL}$  should receive followup as described below.

### ***Followup of children with blood lead levels $\geq 15$ $\mu\text{g/dL}$***

Followup of children with blood lead levels  $\geq 15$   $\mu\text{g/dL}$  is discussed in more detail in later chapters and is briefly summarized below. In general, such children should receive blood lead tests at least every 3 to 4 months.

***If the blood lead level is 15-19  $\mu\text{g/dL}$*** , the child should be screened every 3-4 months, the family should be given education and nutritional counselling as described in Chapter 4, and a detailed environmental history should be taken to identify any obvious sources or pathways of lead exposure. When the venous blood lead level is in this range in two consecutive tests 3-4 months apart, environmental investigation and abatement should be conducted, if resources permit.

***If the blood lead level is  $\geq 20$   $\mu\text{g/dL}$*** , the child should be given a repeat test for confirmation. If the venous blood lead level is confirmed to be  $\geq 20$   $\mu\text{g/dL}$ , the child should be referred for medical evaluation and followup as described in Chapter 7. Such children should continue to receive blood lead tests every 3-4 months or more often if indicated. Children with blood lead levels  $\geq 45$   $\mu\text{g/dL}$  must receive urgent medical and environmental followup, preferably at a clinic with a staff experienced in dealing with this disease. Symptomatic lead poisoning or a venous blood lead concentration  $\geq 70$   $\mu\text{g/dL}$  is a medical emergency, requiring immediate inpatient chelation therapy, as described in Chapter 7.

### **Classification on the Basis of Screening Test Results**

On the basis of screening test results, children can be classified into categories according to their risk for adverse effects of lead. The urgency and type of followup are based on these risk classes. These classes are shown in Table 6-3.

### **Measurement of Blood Lead Levels**

Several factors can influence the quality of blood lead measurements. The ubiquity of lead in the environment makes contamination of specimens during collection a major source of error. Analytical variation in the laboratory can affect results. Accuracy and precision of blood lead measurements, particularly at low concentrations, can be assured by the use of appropriate analytical standards, maintenance of equipment, training of personnel, and participation in external proficiency testing programs.

Since blood collected by venipuncture has a low likelihood of contamination compared to blood collected by fingerstick, venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practicable. In addition, venous specimens provide a larger volume for analysis and are less prone to clotting and other problems that can be encountered with capillary specimens. At the present time, not all laboratories will accept capillary samples for lead analysis.

Fingerstick specimens are acceptable for blood lead screening, provided that special collection procedures are followed to minimize the risk of contamination. Personnel must be thoroughly trained in collection procedures. A procedure for collecting fingerstick specimens is described in Appendix I.

Elevated blood lead results obtained on capillary specimens are presumptive and must be confirmed using venous blood. In general, children who have blood lead levels  $\geq 15$   $\mu\text{g/dL}$  on capillary samples should have these levels confirmed on venous samples, according to the timetable in Table 6-4. A child with a blood lead level  $\geq 70$   $\mu\text{g/dL}$  or with symptoms of lead poisoning should be treated immediately while the results of an immediate confirmatory test are awaited.

**Table 6-3. Class of child and recommended action according to blood lead measurement**

<b>Class</b>	<b>Blood Lead Concentration (<math>\mu\text{g}/\text{dL}</math>)</b>	<b>Action</b>
I	$\leq 9$	Low risk for high-dose exposure: rescreen as described in text. High risk for high-dose exposure: rescreen as described in text.
IIA	10-14	Rescreen as described in text. If many children in the community have blood lead levels $\geq 10$ , community interventions (primary prevention activities) should be considered by appropriate agencies (see Chapter 9).
IIB	15-19	Rescreen as described in text. Take a history to assess possible high-dose sources of lead. Educate parents about diet, cleaning, etc. Test for iron deficiency. Consider environmental investigation and lead hazard abatement if levels persist.
III	20-44*	Conduct a complete medical evaluation. Identify and eliminate environmental lead sources.
IV	45-69*	Begin medical treatment and environmental assessment and remediation within 48 hours.
V	$\geq 70^*$	Begin medical treatment and environmental assessment and remediation IMMEDIATELY.

\*Based on confirmatory blood lead level.

**Table 6-4. Suggested timetable for confirming capillary blood lead results with a venous blood lead measurement**

<b>Blood Lead Level (<math>\mu\text{g}/\text{dL}</math>)</b>	<b>Time Within Which Blood Lead Level Should Be Obtained</b>
<10	Not applicable
10-14	Not applicable
15-19	Within 1 month
20-44	Within 1 week
45-69	Within 48 hours
$\geq 70$	Immediately

### **Blood Lead Levels—Additional Analytical Considerations**

Blood lead levels can be determined by several analytic methods. The method used can affect the specimen volume required, the choice of anticoagulant (usually heparin or ethylenediaminetetraacetic acid (EDTA)), and other aspects related to specimen suitability. Specimen collection procedures and equipment must be checked for compatibility with laboratory

requirements. Special lead-free evacuated tubes are available for blood collection, but standard tubes containing EDTA or heparin (lavender or green caps) can be acceptable after screening each lot to determine the lead content of the containers, needles, etc. Though reports of unsuitable levels of background lead in other collection materials are infrequent, all materials used should be determined to be lead-free before use.

Laboratories where blood is tested for lead levels should be successful participants in a blood lead proficiency testing program, such as the program conducted jointly by CDC, the Health Resources and Services Administration, and the University of Wisconsin. In interpreting laboratory results, it should be recognized that a “proficient” laboratory need only measure blood lead levels to within several  $\mu\text{g}/\text{dL}$  of the true value (for example, within 4 or 6  $\mu\text{g}/\text{dL}$  of a target value). The blood lead level reported by a laboratory, therefore, may be several  $\mu\text{g}/\text{dL}$  higher or lower than the actual blood lead level.

### **Erythrocyte Protoporphyrin (EP)**

EP is not a sensitive test to identify children with blood lead levels below about 25  $\mu\text{g}/\text{dL}$ , and therefore it is no longer the screening test of choice. In some programs, however, it will continue to be used until the transition to blood lead measurements is complete.

Only fresh blood is suitable for analysis by hematofluorometer. Complete oxygenation of sample hemoglobin is necessary to prevent low results in some instruments. The hemoglobin concentration in the sample can also affect hematofluorometer EP readings. Results obtained by extraction methods are not affected by these factors and can be used to confirm hematofluorometer EP results.

In the past, an absorptivity of 241  $\text{L cm}^{-1} \text{mmol}^{-1}$  has been used to determine EP levels. Recently, however, the correct absorptivity has been determined to be 297  $\text{L cm}^{-1} \text{mmol}^{-1}$ . Use of the correct absorptivity will result in EP values about 19% lower than those standardized using 241  $\text{L cm}^{-1} \text{mmol}^{-1}$ . Standardization of EP levels that are based on the correct absorptivity is expected to be widely adopted in 1992. Use of the correct standardization requires a change in calibration and is not simply a reduction of the screening cutoff value. Standardization criteria should also be considered when reviewing data in the literature.

An EP result of  $\geq 35 \mu\text{g}/\text{dL}$  standardized using 241  $\text{L cm}^{-1} \text{mmol}^{-1}$  or  $\geq 28 \mu\text{g}/\text{dL}$  standardized using 297  $\text{L cm}^{-1} \text{mmol}^{-1}$  is considered elevated. **All elevated EP results should be followed with a venous blood lead test to determine if lead poisoning is responsible for the elevation.** Elevated concentrations of EP also result from several health conditions other than lead intoxication, particularly iron deficiency. The iron status of children with elevated EP levels should always be determined, especially since iron deficiency and lead poisoning often coexist. In such cases, the EP may be disproportionately elevated in comparison to the blood lead level.

Some hematofluorometers report EP levels as  $\mu\text{mol ZnPP}/\text{mol heme}$ . For instruments that give results in these units, EP values  $\geq 70 \mu\text{mol}/\text{mol}$  should be considered elevated and should be promptly investigated.

## CHAPTER 7. DIAGNOSTIC EVALUATION AND MEDICAL MANAGEMENT OF CHILDREN WITH BLOOD LEAD LEVELS $\geq 20$ $\mu\text{g/dL}$

Children with blood lead levels between 10  $\mu\text{g/dL}$  and 19  $\mu\text{g/dL}$  and their siblings need followup and repeat screening as described in previous chapters. They do not, however, need medical evaluation as described in this chapter.

The cornerstones of clinical management are careful clinical and laboratory surveillance of the child, medical treatment when indicated, and eradication of controllable sources of environmental lead. **The most important factor in case management is to drastically reduce the child's exposure to lead.**

All children with confirmed venous blood lead levels  $\geq 20$   $\mu\text{g/dL}$  require medical evaluation. The urgency of further medical evaluation depends on the blood lead level and whether symptoms are present.

The decision to institute medical management should virtually always be made on the basis of a venous blood lead measurement. No other screening test can be considered diagnostic. If the first evaluation was made on capillary blood, a confirmatory venous blood lead level must be done. Even if the first diagnostic measurement was on venous blood, it is preferable to retest before starting chelation therapy. For children with blood lead levels  $\geq 70$   $\mu\text{g/dL}$  or clinical symptoms of lead poisoning, chelation should not be postponed while awaiting results of the repeat test.

### Symptoms of Lead Poisoning

**Symptoms of lead poisoning in a child with an elevated blood lead level constitute a medical emergency, and the child should be hospitalized.** Symptoms, which can mimic several other pediatric disorders, must be looked for so they are not missed.

Acute lead encephalopathy is characterized by some or all of these symptoms: coma, seizures, bizarre behavior, ataxia, apathy, incoordination, vomiting, alteration in the state of consciousness, and subtle loss of recently acquired skills. Any one or a mixture of these symptoms, associated with an elevated blood lead level, is an acute medical emergency. Lead encephalopathy is almost always associated with a blood lead level exceeding 100  $\mu\text{g/dL}$ , although, occasionally, it has been reported at blood lead levels as low as 70  $\mu\text{g/dL}$ . Even when identified and promptly treated, severe and permanent brain damage may result in 70%-80% of children with lead encephalopathy. Children with symptomatic lead poisoning with or without encephalopathy represent an acute medical emergency. **The possibility of lead encephalopathy should be considered in the differential diagnosis of children presenting with coma and convulsions of unknown etiology.**

Except for coma and seizures, symptomatic lead poisoning without encephalopathy is characterized by symptoms similar to those of lead encephalopathy. Symptomatic lead poisoning without encephalopathy is characterized by one or a combination of these symptoms: decrease in play activity, lethargy, anorexia, sporadic vomiting, intermittent abdominal pain, and constipation. These symptoms are usually associated with a blood lead levels of at least 70  $\mu\text{g/dL}$ , although occasionally cases have been associated with levels as low as 50  $\mu\text{g/dL}$ . If the blood lead level is below 50  $\mu\text{g/dL}$ , other causes of the symptoms should be sought. **Since acute lead encephalopathy may develop in any symptomatic child, treatment and supportive measures must be started immediately on an emergency basis.**

## Evaluation of the Child with a Blood Lead Level $\geq 20$ $\mu\text{g/dL}$

A child with a blood lead level  $\geq 20$   $\mu\text{g/dL}$  should have a pediatric evaluation, whether or not symptoms are present.

Special attention should be given to:

1. A detailed history, including the presence or absence of clinical symptoms, child's mouthing activities, the existence of pica, nutritional status (especially iron and calcium intake), dietary habits, family history of lead poisoning, potential sources of lead exposure (including exposure due to home renovation), and previous blood lead measurements.
2. Detailed environmental and occupational histories of adults in the household or other places the child spends a lot of time.
3. The physical examination, with particular attention to the neurologic examination and psychosocial and language development. A neurobehavioral assessment may be useful in children receiving chelation therapy both at the time of diagnosis and as the child approaches school age. Findings of language delay or other problems can prompt referral to appropriate programs.
4. Evaluation of iron status using measurement of iron and total iron binding capacity or ferritin.

### Tests

**1. Tests for Iron Deficiency.** *Because iron deficiency can enhance lead absorption and toxicity and often coexists with it, all children with blood lead levels  $\geq 20$   $\mu\text{g/dL}$  should be tested for iron deficiency.* Measurements of hemoglobin, hematocrit, and reticulocytes are not adequately sensitive, and erythrocyte protoporphyrin (EP) is not specific enough to diagnose iron deficiency (although EP can be used to screen for iron deficiency).

**Serum iron and iron binding capacity (transferrin saturation) and ferritin** are the most sensitive indicators of iron status. An abnormally low ratio of serum iron to iron binding capacity (transferrin saturation) of 0.2 is consistent with iron deficiency. The serum ferritin level, however, is the most definitive and accurate indication of overall iron status, although it is an acute phase reactant and may be falsely elevated in sick children; a value  $\leq 12$   $\mu\text{g/dL}$  indicates iron deficiency. Although all iron deficient children should receive treatment for this condition, the treatment should not be started until after chelation is completed in children receiving dimercaprol (BAL).

**2. EP Level.** An elevated EP level indicates impairment of the heme biosynthetic pathway. EP levels are sensitive screening tests for iron deficiency, and iron status should be assessed in any child with an elevated EP level (that is,  $\geq 35$   $\mu\text{g/dL}$  when standardized using 241 L  $\text{cm}^{-1}$   $\text{mmol}^{-1}$ ,  $\geq 28$   $\mu\text{g/dL}$  when standardized using 297 L  $\text{cm}^{-1}$   $\text{mmol}^{-1}$ , or  $\geq 70$   $\mu\text{mol/mol}$  when measured in  $\mu\text{mol/mol}$  units).

Because EP levels take about 2 weeks to increase, EP levels may provide an indication of the duration of lead exposure. Similarly, monitoring the EP level after medical and environmental interventions for poisoned children may be useful. If exposure to lead has ceased, EP values elevated because of lead poisoning decline slowly over several weeks or months. A progressive decline in EP concentrations indicates that combined medical and environmental case management is proceeding efficaciously.

**3. Edetate Disodium Calcium (CaNa<sub>2</sub>EDTA) Provocative Chelation Test.** The mobilization test is used to determine whether a child with an initial confirmatory blood lead level of 25 to 44 µg/dL will respond to chelation therapy with a brisk lead diuresis. Because of the cost and staff time needed for quantitative urine collection, this test is used only in selected medical centers where large numbers of lead-poisoned children are treated. Children whose blood lead levels are ≥45 µg/dL should not receive a provocative chelation test; they should be referred for appropriate chelation therapy immediately.

The outcome of the provocative chelation test is determined not by a decrease in the blood lead level but by the amount of lead excreted per dose of CaNa<sub>2</sub>EDTA given. This ratio correlates well with blood lead levels. In one study, almost all children with blood lead levels 45 µg/dL had positive provocative tests, 76% of the children with blood lead levels 35 to 44 µg/dL had positive test results, and 35% of the children with blood lead levels 25 to 34 µg/dL had positive test results. This test should not be done until the child is iron replete, since iron status may affect the outcome of the test. Details on how to conduct and interpret a provocative chelation test are in Chapter 7.

**4. Radiologic Examination of the Abdomen.** Radiologic examination of the abdomen (flat plate) may show radiopaque foreign material if the material has been ingested during the preceding 24 to 36 hours. Neither negative nor positive xray results are diagnostic or definitive. A flat plate of the abdomen may, however, provide information about the source of lead if paint chips or other lead objects are found.

**5. Radiologic Examination of the Long Bones.** Xrays of the long bones are unreliable for diagnosing acute lead poisoning, and they should not be obtained on a routine basis. They may provide some indication of whether lead poisoning has occurred in the past or has been ongoing for a length of time, and this may occasionally be important. Lines of increased density in the metaphyseal plate of the distal femur, proximal tibia, and fibula may be caused by lead which has disrupted the metabolism of bone matrix. Although these lines are sometimes called lead lines, they are areas of increased mineralization or calcification and not xray shadows of deposited lead.

The following tests are **NOT** indicated for the diagnosis or clinical management of lead poisoning:

**1. Microscopic examination of red cells for basophilic stippling.** Since basophilic stippling is not always found in severe lead poisoning and is insensitive to lesser degrees of lead poisoning, it is not useful in diagnosis.

**2. Tests of hair and fingernails for lead levels.** The levels of lead in hair or fingernails do not correlate well with blood lead levels levels, except in extreme cases of symptomatic lead poisoning; therefore, these tests are not useful in diagnosis. Children should never receive chelating agents on the basis of analyses of lead levels in hair or fingernails.

## **Pharmacology of Chelating Agents**

Several drugs are used in the treatment of lead poisoning. These drugs, capable of binding or chelating lead, deplete the soft and hard (skeletal) tissues of lead and thus reduce its acute toxicity. All drugs have potential side effects and must be used with caution. The basic pharmacologic characteristics of the various drugs are described below.

Chelating Agents Used In Treating Children With Lead Poisoning			
Product Name	Generic Name	Chemical Name	Abbreviation
Calcium Disodium Versenate	Edetate disodium calcium	Calcium disodium ethylenediamine tetraacetate	CaNa <sub>2</sub> EDTA
BAL in Oil	Dimercaprol	2,3-dimercapto-1-propanol	BAL
Cuprimine	D-penicillamine	3-mercapto-D-valine	D-penicillamine
Chemet	Succimer	Meso 2,3-dimercaptosuccinic acid	DMSA

### **BAL**

**Mechanism of action.** Two molecules of dimercaprol (BAL) combine with one atom of heavy metal to form a stable complex. BAL enhances fecal and urinary excretion of lead and diffuses well into erythrocytes. Because it is predominantly excreted in bile, BAL can be administered in the presence of renal impairment.

**Route of administration and dosage.** BAL is available only in peanut oil for intramuscular administration. It is usually given every 4 hours, although it may be given every 8 hours; dosages are discussed below.

**Precautions and Toxicity.** For patients with glucose-6-phosphate dehydrogenase deficiency (G-6-PD), some clinicians recommend that BAL should be used only in life-threatening situations because it may induce hemolysis. Medicinal iron should never be administered during BAL therapy, because the combination of iron and BAL has been implicated in serious reactions. If iron deficiency coexists, it should not be treated until after BAL therapy has been completed. In cases of extreme anemia, blood transfusions are preferable.

Between 30% and 50% of patients who receive BAL will experience side effects. Mild febrile reactions and transient elevations of hepatic transaminases may be observed. Other minor adverse effects include, in order of frequency, nausea and occasional vomiting, headache, mild conjunctivitis, lacrimation, rhinorrhea, and salivation. Most side effects are transient and rapidly subside as the drug is metabolized and excreted. Intravenous hydration coupled with restricting oral intake can circumvent, in large part, gastrointestinal distress. **BAL should not be used for children who are allergic to peanuts or peanut products.**

### **CaNa<sub>2</sub>EDTA**

*Only CaNa<sub>2</sub>EDTA can be used for treating children with lead poisoning. Na<sub>2</sub>EDTA (disodium edetate) should never be used for treating children with lead poisoning because it will induce tetany and possibly fatal hypocalcemia.*

**Mechanism of action.** CaNa<sub>2</sub>EDTA increases urinary lead excretion twentyfold to fiftyfold. CaNa<sub>2</sub>EDTA removes lead from the extracellular compartment only, because it does not enter cells.

**Route of administration and dosage.** The preferred route for administration of CaNa<sub>2</sub>EDTA is intravenous. CaNa<sub>2</sub>EDTA must be diluted to a concentration <0.5% in dextrose and water or in 0.9% saline solution. It can be given as a continuous infusion or it can be given in two divided doses a day through a heparin lock over 30 to 60 minutes. CaNa<sub>2</sub>EDTA causes

extreme pain when administered intramuscularly; therefore, when given by this route, it should be mixed with procaine so that the final concentration of procaine is 0.5%.  $\text{CaNa}_2\text{EDTA}$  should never be given orally because it enhances absorption of lead from the gastrointestinal tract.

Dosages vary by situation and are detailed in Chapter 7. Individual courses should be limited to 5 days and repeated courses should be given at a minimum of 2- to 5-day intervals. Particularly when  $\text{CaNa}_2\text{EDTA}$  is given on an outpatient basis, some clinicians use sequential 3-day courses of treatment.

**Precautions and Toxicity.** During chelation therapy with  $\text{CaNa}_2\text{EDTA}$ , urine output, urine sediment, blood urea nitrogen (BUN), serum creatinine, and hepatocellular enzyme levels must be carefully monitored. The appearance of protein and formed elements in urinary sediment, and rising BUN and serum creatinine values reflect impending renal failure—the serious toxicity associated with inappropriately excessive or prolonged administration of  $\text{CaNa}_2\text{EDTA}$ . Liver transaminases may increase by the fifth day of therapy, but return to pretreatment levels within a week after treatment has ended.

When  $\text{CaNa}_2\text{EDTA}$  is used alone without concomitant BAL therapy, it may aggravate symptoms in patients with very high blood lead levels. Therefore, it should be used in conjunction with BAL when the blood lead level is  $\geq 70 \mu\text{g/dL}$  or overt clinical symptoms of lead poisoning are present. In such cases, the first dose of BAL should always precede the first dose of  $\text{CaNa}_2\text{EDTA}$  by at least 4 hours.

The kidney is the principal site of potential toxicity. Renal toxicity is dose related, reversible, and rarely (if ever) occurs at doses  $< 1500 \text{ mg/m}^2$  when the patient is adequately hydrated.  $\text{CaNa}_2\text{EDTA}$  must never be given in the absence of an adequate urine flow.

### ***D-penicillamine***

The Food and Drug Administration (FDA) has approved D-penicillamine for the treatment of Wilson's disease, cystinuria, and severe, active rheumatoid arthritis. Although not approved for this use, it is used in some centers for treating lead poisoning. Until the recent approval of succimer, it was the only commercially available oral chelating agent. It can be given over a long period (weeks to months). D-penicillamine has been used mainly for children with blood lead levels  $< 45 \mu\text{g/dL}$ .

**Mechanism of action.** D-penicillamine enhances urinary excretion of lead, although not as effectively as  $\text{CaNa}_2\text{EDTA}$ . Its specific mechanism and site of action are not well understood.

**Route of administration and dosage.** D-penicillamine is administered orally. It is available in capsules or tablets (125 mg and 250 mg). These capsules can be opened and suspended in liquid, if necessary. The usual dose is 25 to 35 mg/kg/day in divided doses. Side effects can be minimized, to an extent, by starting with a small dose and increasing it gradually, monitoring all the time for side effects. For example, 25% of the desired final dose could be given in week 1, 50% in week 2, and the full dose by week 3.

**Precautions and Toxicity.** Toxic side effects (albeit minor in most cases) occur in as many as 33% of patients given the drug. The main side effects of D-penicillamine are reactions resembling those of penicillin sensitivity, including rashes, leukopenia, thrombocytopenia, hematuria, proteinuria and hepatocellular enzyme elevations, and eosinophilia. Anorexia, nausea, and vomiting are infrequent. Of most concern, however, are isolated reports of nephrotoxicity, possibly from hypersensitivity reactions. For these reasons, patients should be carefully and frequently monitored for clinically obvious side effects, and frequent blood counts,

urinalyses, and renal function tests should be performed. In particular, blood counts and urinalyses should be done on day 1, day 14, day 28, and monthly thereafter. If the absolute neutrophil count falls to  $<1500/\mu\text{L}$ , the count should be rechecked immediately, and treatment should be stopped if it falls to  $<1200/\mu\text{L}$ . D-penicillamine should not be given on an outpatient basis if exposure to lead is continuing or the physician has doubts about compliance with the therapeutic regimen. **D-penicillamine should not be administered to patients with known penicillin allergy.**

### **Succimer**

The FDA approved succimer in January, 1991 for treating children with blood lead levels  $>45 \mu\text{g/dL}$ . Succimer appears to be an effective oral chelating agent. Its selectivity for lead is high, whereas its ability to chelate essential trace metals is low. Although its use to date has been limited, succimer appears to have promising potential, and a broader range of clinical research studies in children are being undertaken.

Succimer is chemically similar to BAL but is more water soluble, has a high therapeutic index, and is absorbed from the gastrointestinal tract. It is effective when given orally and produces a lead diuresis comparable to that produced by  $\text{CaNa}_2\text{EDTA}$ . This diuresis lowers blood lead levels and reverses the biochemical toxicity of lead, as indicated by normalization of circulating aminolevulinic acid dehydrase levels. Succimer is not indicated for prophylaxis of lead poisoning in a lead-containing environment. **As with all chelating agents, succimer should only be given to children who reside in environments free of lead during and after treatment.**

**Mechanism of Action.** Succimer appears to be more specific for lead than the most commonly used chelating agent,  $\text{CaNa}_2\text{EDTA}$ ; the urinary loss of essential trace elements (for example, zinc) appears to be considerably less with succimer than with  $\text{CaNa}_2\text{EDTA}$ . The site of lead chelation by succimer is not known.

**Route of Administration and Dosage.** Succimer is administered orally. It is available in 100 mg capsules. The recommended initial dose is  $350 \text{ mg/m}^2$  ( $10 \text{ mg/kg}$ ) every 8 hours for 5 days, followed by  $350 \text{ mg/m}^2$  ( $10 \text{ mg/kg}$ ) every 12 hours for 14 days. A course of treatment, therefore, lasts 19 days. If more courses are needed, a minimum of 2 weeks between courses is preferred, unless blood lead levels indicate the need for immediate retreatment. These doses may be modified as more experience is gained in using succimer.

Patients who have received therapeutic courses of  $\text{CaNa}_2\text{EDTA}$  with or without BAL may use succimer for subsequent treatment after an interval of 4 weeks. Data on the concomitant use of succimer and  $\text{CaNa}_2\text{EDTA}$  with or without BAL are not available, and such use is not recommended.

If young children cannot swallow capsules, succimer can be administered by separating the capsule and sprinkling the medicated beads on a small amount of soft food or by putting them on a spoon and following with a fruit drink. Data are not available on how stable succimer is when it is suspended in soft foods for prolonged periods of time; succimer should be mixed with soft foods immediately before being given to the child.

**Precautions and Toxicity.** To date, toxicity due to succimer (transient elevations in hepatic enzyme activities) appears to be minimal. The most common adverse effects reported in clinical trials in children and adults were primarily gastrointestinal and included nausea, vomiting, diarrhea, and appetite loss. Rashes, some necessitating discontinuation of therapy, have been reported for about 4% of patients. **Though succimer holds considerable promise for the**

**outpatient management of lead poisoning, clinical experience with succimer is limited.** Consequently, the full spectrum and incidence of adverse reactions, including the possibility of hypersensitivity or idiosyncratic reactions, have not been determined. Other precautions that need to be taken with succimer are discussed in the full statement.

### **Treatment Guidelines For Children With Blood Lead Levels $\geq 20$ $\mu\text{g/dL}$**

**The single most important factor in managing of childhood lead poisoning is the reducing the child's exposure to lead; some children, however, will benefit from chelation therapy.** Sample regimens for treating children with lead poisoning are described in Chapter 7.

#### ***Medical Management of Symptomatic Lead Poisoning (with or without Encephalopathy)***

Children with symptomatic lead poisoning (with or without encephalopathy) must be treated only at a pediatric center that has an intensive care unit. They should be managed by a multidisciplinary team that includes, as needed, critical care, toxicology, neurology, and neurosurgery. The child's neurological status and fluid balance must be carefully monitored.

#### ***Medical Management of Asymptomatic Lead Poisoning***

**Blood lead level  $\geq 45$   $\mu\text{g/dL}$ .** Children with blood lead levels  $\geq 45$   $\mu\text{g/dL}$  (with or without symptoms) should undergo chelation therapy. A blood lead level  $\geq 70$   $\mu\text{g/dL}$  is a medical emergency.

**Blood lead level 25 to 44  $\mu\text{g/dL}$ .** For this blood lead range, the effectiveness of chelation therapy in decreasing the adverse effects of lead on children's intelligence has not been shown. Treatment regimens vary from clinic to clinic. Some practitioners treat children with lead levels in this range pharmacologically, some use D-penicillamine. The minimum medical management for children with these blood lead levels is to decrease exposure to all sources of lead, to correct any iron deficiency and maintain an adequate calcium intake, and to test frequently to ensure that the child's blood lead levels are decreasing. Many experienced practitioners decide whether to use chelation therapy on the basis of the results of carefully performed  $\text{CaNa}_2\text{EDTA}$  mobilization tests.

**Blood lead level 20 to 24  $\mu\text{g/dL}$ .** Only very minimal data exists about chelating children with blood lead levels below 25  $\mu\text{g/dL}$ , and such children should not be chelated except in the context of approved clinical trials. A child with a confirmed blood lead level of 20 to 24  $\mu\text{g/dL}$  will require individual case management by a pediatric health-care provider. The child should have an evaluation with special attention to nutritional and iron status. The parents should be taught about 1) the causes and effects of lead poisoning, 2) the need for more routine blood lead testing, 3) possible sources of lead intake and how to reduce them, 4) the importance of adequate nutrition and of foods high in iron and calcium, and 5) resources for further information. (This is described in more detail in Chapter 4.) Sequential measurements of blood lead levels along with review of the child's clinical status should be done at least every 3 months. Iron deficiency should be treated promptly. Children with blood lead levels in this range should be referred for environmental investigation and management. **Identifying and eradicating all sources of excessive lead exposure is the most important intervention for decreasing blood lead levels (Chapter 8).**

## Post-Chelation Followup

At the end of each treatment cycle, the blood lead concentration usually declines to  $<25$   $\mu\text{g/dL}$ . Within a few days, however, reequilibration among body lead compartments takes place and may result in a rebound; thus, **the blood lead level must be rechecked 7 to 21 days after treatment to determine whether retreatment is necessary.**

Children who undergo chelation treatment require long-term followup preferably from pediatric health-care providers, nutritionists, environmental specialists, and community outreach workers. Community outreach workers provide a critical bridge between hospital-based or clinic-based (outpatient) medical care, health advocacy education, and environmental remediation outside the hospital. Children should **never** be discharged from the hospital **until they can go to a lead-free environment.** Lead-free safe housing (with friends, relatives, or in designated transitional housing), in which a treated child can live during the entire abatement process through the post-abatement clean-up, must be arranged. With appropriately carried-out public health measures, complete and safe abatement should be achieved during the treatment period.

Once a child is discharged to a safe environment, frequent followup is mandatory. In general, depending on the initial blood lead value, most children who require chelation therapy must be followed closely for at least one year or more. All children undergoing chelation treatment should be seen every other week for 6-8 weeks, then once a month for 4-6 months. A child treated with BAL and  $\text{CaNa}_2\text{EDTA}$  should be followed more closely: weekly for 4 to 6 weeks, then monthly for 12 months.

## CHAPTER 8. MANAGEMENT OF LEAD HAZARDS IN THE ENVIRONMENT OF THE INDIVIDUAL CHILD

Eradicating childhood lead poisoning requires a long-term active program of primary lead-poisoning prevention, including abatement of lead-based paint hazards in homes, day-care centers, and other places where young children play and live. For the child who is lead poisoned, however, efficient and effective interventions are needed as quickly as possible. Abatement means making the source of lead inaccessible to the child.

Each situation in which a child gets poisoned is unique and must be evaluated by a person or team of persons skilled and knowledgeable about lead poisoning, hazard identification, and interventions to reduce lead exposure, including abatement of lead-based paint in housing. Childhood lead poisoning prevention programs need to work closely with other relevant agencies (for example, housing and environmental agencies) to ensure that the quickest and most effective approach is taken to remediating the environments of poisoned children.

Environmental case management includes a number of actions prescribed for a child with lead poisoning. Ideally, environmental case management should be conducted by a team of professionals in public health, environmental activities, medical management, and social management. A team approach to intervention will help ensure that followup is timely and effective. The management team may need to solve many related problems, such as whether to investigate supplemental addresses, where to find temporary alternative housing, and how to use community resources to assist the family in dealing with the lead-poisoned child.

## **CHAPTER 9. MANAGEMENT OF LEAD HAZARDS IN THE COMMUNITY**

In theory, primary prevention has always been the goal of childhood lead poisoning prevention programs. In practice, however, most programs focus exclusively on secondary prevention, dealing with children who have already been poisoned. As programs shift the emphasis to primary prevention, their efforts must be designed to systematically identify and remediate environmental sources of lead, including, most importantly, dwellings containing old lead paint.

The shift from case management to community-level intervention will require a fundamental shift in perspective. The focus must shift from the individual child to the population of children at risk and the environment in which they live. The purpose of community-level intervention is to identify and respond to sources, not cases, of lead poisoning. The responsibility for addressing lead poisoning will have to be expanded beyond health agencies to include a variety of housing, environmental, and social service agencies at the local, county, state, and national level.

# Notes